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Original Communications

Department of Clinical Reports

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Original Communications

IMMUNIZATION AGAINST RHEUMATIC FEVER WITH HEMOLYTIC STREPTOCOCCUS FILTRATE*

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THIS paper discusses an attempt to immunize 34 children who have had one or more attacks of rheumatic fever with stock filtrate of *Streptococcus hemolyticus*. The procedure was suggested by a similar method used by Dr. Franklin Stevens in treating bacterial asthma and arthritis in adults at the Presbyterian Hospital.

We realize that the number of patients treated is too small to be able to draw definitive conclusions, but the results appear significant enough for a preliminary report, in the hope of stimulating research along similar lines.

The treated patients, designated here as Group A, are divided into two groups. Ten children received inoculations with the filtrate from September 1933 to June 1935 (Group A1),† and 24 other children from September 1935 to June 1937 (Group A2).

In each instance 34 patients were used as controls (Groups B1 and B2). These controls were examined regularly in the rheumatic fever and cardiac clinics and received standard treatment as complaints arose, but no attempt was made to immunize them.

According to Stevens‡ it requires approximately two years to treat a case of bacterial asthma. We decided to apply the same tentative rule to patients with rheumatic fever.

Preparation of the Filtrate.—Stock filtrate only was used. A strain of scarlatinal streptococcus N.Y.5 was obtained through the courtesy of Dr. Franklin Stevens and was used exclusively in the preparation of the material. The bacteria were grown for four days in 50 c.c. of 2 per cent proteose peptone broth. The material was filtered; the filtrate was tested for sterility, and four normal saline dilutions were set up: 1:100, 1:50, 1:20, and 1:5. Some undiluted filtrate was kept for the largest doses.

*From the Department of Pediatrics, Fifth Avenue Hospital, 1933-35, and the Department of Cardiology, Flower-Fifth Avenue Hospital, 1935-37.

†Reported before the Pediatric Section of the Medical Society of the County of New York, March, 1936.

‡Personal communication, 1933.

Mode of Administration.—Subcutaneous injections were first given weekly in graduated doses of 0.3, 0.6, 0.9 c.c. beginning with the 1:100 dilution, followed by similar doses of stronger concentrations. After 12 injections, a month was allowed to elapse, at the end of which time 0.3 c.c. of the full strength filtrate was given, followed at monthly intervals by 0.6 and 0.9 c.c. The latter dose was repeated monthly until the beginning of June. The treatment was then discontinued until September when the whole course was repeated as before. No more than a local reaction with the larger doses was ever observed.

CHOICE OF PATIENTS

The only criterion used for selecting the patients for treatment was their willingness to cooperate during the protracted course of inoculations, involving weekly and monthly visits to the clinic. Age, sex, and the severity of the disease played no part in our choice.

The control Group B consisted either of patients whose parents for some reason could not or would not bring them to the clinic frequently and regularly, or of those who joined the clinic too late to receive the full course of treatment. On the whole the cooperation of the families was remarkably good, and only two patients in the control Group B2 disappeared in the course of two years.

Half of the patients were Italian, one-quarter Puerto Ricans, and the rest were mostly Irish, Jewish, or American Negro. The economic and educational status of the majority was low.

ROUTINE PROCEDURE AT THE CLINIC

To evaluate the results of the therapy it is necessary to know about the routine "work-up" of each patient. On admission a detailed history was taken consisting of past, rheumatic, family, racial, and hygienic histories. A careful inquiry was made into the child's habits and mental and emotional make-up.

Each patient was then tested with hemolytic streptococcus nucleoprotein to determine his sensitivity to the organism, followed by a blood count, erythrocyte sedimentation rate, and throat culture for predominating organisms. The parents received printed instructions in English, Spanish, or Italian telling how to deal with a child with rheumatic fever. Each patient was examined regularly at the heart clinic. The assistance of the social service department was invoked whenever necessary.

At each subsequent visit the interval history was taken, a throat culture and sedimentation rate done, and a blood count as often as possible. Whenever subacute rheumatic symptoms or an upper respiratory infection presented itself the child was sent home to bed and was reexamined a week later. In case of a frank attack of rheumatic fever the patient was often admitted to the ward. Convalescent care was arranged for as many children as possible.

DISCUSSION OF RESULTS

For clarity's sake we will first compare the results in Group A1, comprising 10 children treated over the period of two years, from September 1933 to June 1935, with its control group of 34 patients, designated as B1, and then the group of 24 children, called Group A2, whose treatment lasted from September 1935 to June 1937, with its corresponding control Group B2.

Table I gives the comparison between Groups A1 and B1 before the treatment was instituted, and Table II does the same for Groups A2 and B2. Tables III and IV represent the findings in the treated and the control groups respectively during each two-year course of immunization. In Tables V and VI we place under "results" our estimate of the child's physical condition from the general and the rheumatic fever point of view at the end of each two years of therapy.

TABLE I

COMPARISON OF GROUPS A1 AND B1 BEFORE TREATMENT

	TREATED GROUP	CONTROL GROUP
No. of patients	10	34
Average age	8.7 yr.	9.47 yrs.
Sex	Males 5, Females 5	Males 11, Females 23
Cardiac class number	F, 2; E+F, 5; I, 1; IIa, 1; IIb, 1	F, 9; E+F, 7; I, 9; IIa, 7; IIb, 2
History of acute rheumatic fever	7, 70 per cent	19, 56 per cent
Positive skin test	10, 100 per cent	27, 79.5 per cent

TABLE II

COMPARISON OF GROUPS A2 AND B2 BEFORE TREATMENT

	TREATED GROUP	CONTROL GROUP
No. of patients	24	32
Average age	7.2 yr.	8.5 yr.
Sex	Males 11, Females 13	Males 15, Females 17
Cardiac class number	F, 9; E+F, 7; I, 3; IIa, 5	F, 8; E+F, 12; I, 7; IIa, 4; IIb, 1
History of acute rheumatic fever	All	All
Positive skin test	19, 70.9 per cent	28, 87.5 per cent

Group A1.—Ten children are included in this group. Their average age was 8.7 years at the time the treatment was begun in September 1933. The sexes were equally divided. Two children had severe heart disease (Cardiac Class II). In the course of treatment during the following two years the average erythrocyte sedimentation rate* and hemoglobin percentage were 10.5 mm. and 75 per cent, respectively. As far as infections are concerned there has not been a single instance

*Landou's microsedimentation method was used exclusively on ambulatory patients. The normal findings are up to 8 mm. per hour.

TABLE III

COMPARISON OF GROUPS A1 AND B1 DURING TREATMENT SEPTEMBER 1933–JUNE 1935

	TREATED GROUP	CONTROL GROUP
Average erythrocyte sedimentation rate	10 mm. per hr.	13 mm. per hr.
Average hemoglobin per cent	75	72
Total number of colds	28	77
No. of colds per child	2.8	2.57
No. of rheumatic fever attacks	0	7 in 5 patients
Hospitalization	2	15
Convalescent care	2	3
Pallor	3+,* 1+++	7+, 7++, 9+++
Abdominal symptoms	4+, 1++, 1+++	9+, 3++, 9+++
Headaches	1+++	8+, 6++, 6+++
Epistaxis	2+	5+, 6++, 4+++
Rheumatic pains	3+, 2++, 1+++	6+, 8++, 9+++
Average gain in weight	15.5 lb.	6.82 lb.

* +, occurred once
++, occurred twice
+++, occurred many times

TABLE IV

COMPARISON OF GROUPS A2 AND B2 DURING TREATMENT SEPTEMBER, 1935—JUNE, 1937

	TREATED GROUP	CONTROL GROUP
Average erythrocyte sedimentation rate	8.5 mm. per hr.	11 mm. per hr.
Average hemoglobin per cent	80	76
Total number of colds	103 for 20 patients	130 for 23 patients
Chronic sinusitis	4, at the end 2	9
Average number of colds	4.2	5.6
Number of rheumatic fever attacks	2	17 in 14 patients
Hospitalization	1	7
Pallor	3+,* 2++, 4+++	5+, 3++, 10+++
Abdominal symptoms	9+, 3++, 2+++	8+, 10++, 9+++
Headaches	2+, 5++, 4+++	5+, 5++, 18+++
Epistaxis	2+, 4++, 1+++	2+, 4++, 9+++
Joint and muscle pains	7+, 4++	3+, 6++, 18+++
Average gain in weight	10.9 lb.	10.5 lb.

* +, occurred once
++, occurred twice
+++, occurred many times

TABLE V

	GROUP A1 RESULTS OF TREATMENT		GROUP B1 CONTROL	
	10 cases		34 cases	
Very good	9	90 per cent	5	14.7 per cent
Good	1	10 per cent	9	23.5 per cent
Fair	0	0	10	25.9 per cent
Poor	0	0	10	25.9 per cent

TABLE VI

	GROUP A2 RESULTS OF TREATMENT		GROUP B2 CONTROL	
	24 cases		32 cases	
Very good	17	70 per cent	5	15.8 per cent
Good	4	17 per cent	10	31 per cent
Fair	2	8.9 per cent	5	15.8 per cent
Poor	1	4.1 per cent	12	37.4 per cent

of acute rheumatic fever during the entire course of treatment and there were 2.8 other infections per child, including colds, a case of lobar pneumonia, and a case of otitis media. The average gain in weight per child for two years was 15.5 pounds.

Abdominal symptoms were almost absent. Only one child during the first year complained of frequent abdominal pain. Pallor was observed in four patients and was marked in one. One child complained of headaches which disappeared as soon as an old sinus infection was effectively treated in the ward. Three patients reported joint pains on one transient occasion, two on two occasions and one frequently. No twitching or nervousness was observed in this group. There has not been a single hospital admission for rheumatic fever, but there was one for pneumonia and one for sinusitis. Two children received convalescent care.

Group B1.—Thirty-four children were included in this group. They were all registered in the clinic on an average for thirteen months. The mean age at the time of enrollment was 9.57 years. Eleven of the patients were boys and 23 girls. Nine children, or 25 per cent, were in the cardiac Class II, i.e., they had severe heart disease. Nineteen children (56 per cent) had a history of one or more attacks of rheumatic fever, and seven had had chorea (all with one exception in combination with other rheumatic equivalents), and the remainder of the eight children gave a history of rheumatic symptoms without definite attacks of rheumatic fever. The average sedimentation rate and hemoglobin percentage for the total time of observation were 13 mm., and 72 per cent respectively. Thirty children reported 77 colds and four had chronic sinusitis and were never free from colds. Excluding the cases with sinusitis this makes 2.57 colds per child. The mean gain in weight per patient was 6.82 pounds.

Abdominal pain and vomiting were reported in 23 patients and in nine of them the symptoms were frequent and severe. Twenty-one children also showed pallor, often to a marked degree, 20 complained of headaches (severe in six), and 15 had epistaxis on one or more occasions. Joint and muscle pains occurred in 23 patients. There were seven attacks of acute rheumatic fever in five children. There were 15 admissions to the hospital in this group and three patients were sent to the country for convalescent care.

Group A2.—The results of the first two years of treatment were so encouraging that in September 1935 we began to immunize a new

group of 24 children. The control Group B2 was made up of 14 patients who were studied for the two preceding years in control Group B1 and 20 new patients. By the end of the first year two children had drifted away and hence observations on only 32 control patients are presented here.

In Group A2 there were 11 males and 13 females. Their average age at the beginning of inoculations was 7.2 years. Five patients had severe heart disease. All the children in this group had a definite history of attacks of rheumatic fever.

During the course of treatment the average erythrocyte sedimentation rate and hemoglobin percentage were 7.8 mm. and 80 per cent respectively. The total number of colds was 103 for 20 children during two winters, or 4.2 per child. Four had chronic sinus infection during the first year and only two during the second. There were two attacks of rheumatic fever in two children. One of them was hospitalized for two months, although she showed no cardiac involvement and her white cell count and sedimentation rate were only slightly raised. The other case was diagnosed by a private physician and the boy stayed in bed at home for two weeks.

Indefinite subacute rheumatic symptoms were few in this group. Only four children showed frequent pallor (16.6 per cent), two children complained of abdominal symptoms on several occasions (8.3 per cent), four had frequent headaches (16.6 per cent) and only one had more than two nosebleeds (4.15 per cent). Not a single patient complained more than twice of transitory joint or muscle pains. There has been one admission to the hospital for rheumatic fever and one for pinworm infestation. Four children spent a month in the country. The average gain in weight per child was 10.9 pounds for two years. The five children who at the beginning of therapy were diagnosed as Class IIa cardiacs passed into Class I.

One child reported precordial pain on one occasion and another on two occasions. No other cardiac symptoms were recorded.

Group B2.—Thirty-two control patients were observed for the same period of two years. Their average age was 8.5 years. Fifteen were males and 17 females. Five belonged to cardiac Class II. The average hemoglobin percentage and sedimentation rate for this group were 76.8 and 10.7 mm. There were 130 upper respiratory infections reported by 23 patients and nine had chronic sinusitis throughout the time of observation. The usual treatment in the ear, nose, and throat department brought only transitory relief.

This group fared particularly badly as far as rheumatic fever was concerned. Fourteen patients had 17 attacks during the two consecutive winters. The disease in its acute manifestations affected 43.4 per cent of the entire group.

The subacute rheumatic symptoms were many and troublesome. Ten patients had chronic pallor (31.2 per cent), nine, or 28 per cent, fre-

quent attacks of vomiting and abdominal pain, 18, or 56 per cent, had constantly recurring headaches, many of them due in all probability to chronic sinusitis. Nine children (28 per cent) reported repeated nosebleeds, and 18, or 56 per cent, complained constantly of joint and muscle pains.

The cardiac symptoms were also prominent. Four children complained frequently of palpitation, seven of precordial pain, and ten of dyspnea. There were four hospital admissions for rheumatic fever, one for an appendicectomy, two for otitis media, and seven patients received country care.

The average gain in weight however was good and almost identical with the treated group, namely 10.5 pounds.

Observations on Group A1 for Two Years Following Treatment

It was obviously of interest for us to know how the first treated group has fared since the inoculations were discontinued in June 1935. These patients have reported to the clinic regularly since then and it was easy to estimate their state of health.

Unfortunately one boy died of meningococcus meningitis at another hospital in May 1936. Previous to this he was in excellent health.

Of the other nine children one developed an attack of rheumatic fever with cardiac decompensation in March 1936 and was placed in cardiac Class IIa. He has been sent to a cardiac convalescent home. The remaining eight children have shown a perfect health record. Including the patient who had an attack of rheumatic fever, the average hemoglobin and sedimentation rate were 80 per cent and 9 mm. respectively. There was a total of 18 colds and no sinus infections. The total gain in weight was 150 pounds, or over 17 pounds per child. This record however is misleading, as one girl gained 41 pounds in two years from overeating. Her basal metabolism remained normal. In 1933 before the inoculations were begun, she was a Class IIb cardiac, in 1935 she was classed as IIa, and in 1937 as Class I, and then began to attend school for normal children. Another child passed from Class IIa to Class I.

Electrocardiographic Studies

It was of utmost importance to determine if the administration of hemolytic streptococcus filtrate had any adverse effect upon the myocardium, as might be revealed by a prolongation of the P-R interval or other changes. Electrocardiographic studies were made on each child in Group A2 just before the treatment was begun and at least twice during the course of treatment. No pathological changes were observed then. To determine whether the filtrate had any immediate deleterious effect upon the heart muscle, 20 out of 24 children had an electrocardiogram taken within twenty-four hours of the largest dose of the filtrate.

It will be seen from Table VII that only in one instance (Case 1) was the P-R interval prolonged from 0.19 second with a rate of 120 to 0.14 second at the rate of 150.

TABLE VII

COMPARISON OF P-R INTERVAL IN ELECTROCARDIOGRAMS OF 20 PATIENTS TAKEN BEFORE COMMENCEMENT OF TREATMENT AND WITHIN TWENTY-FOUR HOURS OF THE LARGEST DOSE OF THE HEMOLYTIC STREPTOCOCCUS FILTRATE

NO.	BEFORE TREATMENT		AFTER LAST INJECTION	
	RATE	P-R	RATE	P-R
		(SECOND)		(SECOND)
1.	120-30	0.19	150	0.14
2.	100	0.18	90	0.17
3.	100	0.16	100	0.16
4.	100	0.16	74	0.16
5.	110-20	0.17	130	0.16
6.	100	0.18	100	0.18
7.	90	0.12	96	0.12
8.	96	0.19	110	0.18
9.	100	0.20	100	0.20
10.	110	0.16	110	0.16
11.	76	0.16	80	0.19
12.	100-90	0.20	90	0.20
13.	96	0.18	80	0.19
14.	90-80	0.18	80	0.19
15.	100-90	0.20	120	0.18
16.	90	0.16	86	0.16
17.	110-20	0.14	126	0.16
18.	80	0.20	90	0.20
19.	88	0.19	90	0.20
20.	110	0.16	120	0.12

COMMENT

From the above data it appears that the 34 children who have received inoculations of hemolytic streptococcus filtrate fared remarkably well as compared with the untreated groups and even with the average health experience of any group of normal children. Whether this was due to therapy or merely to a fortunate chance selection of patients cannot be stated dogmatically. We realize that the groups are too small to make definitive conclusions possible, but we cannot help feeling impressed by the low incidence of rheumatic symptoms and attacks of rheumatic fever in the treated group. Out of 34 children only two, or 5.9 per cent, developed the disease in its acute phase during the course of therapy, while the control groups showed 15 per cent and 43.4 per cent respectively. The physical condition of the control patients remained mediocre, and complaints of pains, headaches, and fatigue were frequent.

The purpose of this article is not to draw conclusions, but to state our observations. It is our intention to treat a much larger group of patients during the next two years and make extensive electrocardiographic studies in order to determine the immediate and remote effects of the filtrate on the heart muscle.

EVALUATION OF RESULTS IN TREATMENT OF PERIPHERAL CIRCULATORY DISEASES*

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DURING the past fifteen years there has been a noticeable change in the attitude of the profession toward the prognosis in patients with peripheral vascular diseases. Up to about the year 1925, the prevailing opinion was that little could be done for sufferers from the organic arterial diseases, and that eventually amputation of the extremities was usually necessary. This generally hopeless attitude was a reaction to the poor results of treatment obtained prior to this period.

The last decade has witnessed an extraordinary development of new methods of treatment for patients with peripheral circulatory disorders. Table I presents an incomplete list of the methods proposed and practiced during this time. The growing interest in this subject

TABLE I

METHODS USED IN TREATMENT OF PERIPHERAL VASCULAR DISEASE

- Physiotherapy measures
  - Heat
    - Hot baths
    - Baking
    - Thermo-regulated cradle
    - Diathermy
    - Short wave
  - Whirlpool baths
  - Buerger's exercises
  - X-ray treatment over spine
  - Suction-pressure apparatus (Pavaex)
  - Intermittent venous hyperemia
- Injections
  - Hypertonic sodium chloride
  - Sodium citrate
  - Typhoid vaccine
  - Insulin-free pancreatic tissue extract
  - Paravertebral injections of alcohol
  - Subarachnoid injections of alcohol
  - Sodium thiosulfate
- Drugs
  - Theobromine
  - Acetylcholine
  - Mecholin
  - Nitrites
  - Iodides
  - Papaverine
  - Alcohol
  - Allantoin
  - Thioglycerol
- Operations
  - Perivascular sympathectomy (Leriche)
  - Ganglionectomy
  - Arteriectomy
  - Vein ligation
  - Peripheral nerve section

*From the clinic for Peripheral Vascular Diseases, the Mount Sinai Hospital.

has been reflected in numerous papers dealing with diseases of the peripheral circulation. Enthusiastic reports on the results of treatment by various methods have appeared, indicating a new spirit of optimism. In many of the reports of the good results obtained by different forms of therapy, a critical attitude is noticeably lacking. In numerous acute and chronic ailments it is the spontaneous course of the disease rather than the method of treatment which is responsible for the recovery of the patient. In the treatment of peripheral vascular disease there is too great a readiness to accept improvement as indicating the value of the treatment used. It is important to realize that in these conditions also considerable spontaneous improvement frequently takes place. Other factors, such as the benefit from cessation of smoking and the psychic effect of treatment, are frequently ignored.

Following occlusion of a major artery in an extremity, nature comes to the aid of the patient and quickly develops a collateral circulation. This process of building up a collateral circulation proceeds rapidly during the first year and continues for at least two or three years longer. While treatment may accelerate this process, it will occur even though no treatment is employed.

Figure 1 illustrates diagrammatically the changes in circulation which take place after such a closure. The first period is one of improvement of circulation due to the spontaneous development of collateral circulation. This process continues for two or three years whether or not treatment is used. Following this first period, the circulation remains at a remarkably stationary level for an indeterminate number of years, indicated by the broken line. During this second period the collateral circulation is maintained. In patients with arteriosclerosis there follows a third period when the collateral circulation itself gradually becomes involved by the arteriosclerotic process, causing a progressive diminution in circulation. It is at once apparent that it makes a great difference whether the patient is at point *A*, *B*, or *C* when first seen. If it is claimed that the patient who is at *A* responded well to a form of treatment, it is logical to point out that this patient was improving spontaneously and treatment may have had no influence whatsoever. If it is stated that as a result of treatment of a patient at *B* there was no increase in circulation but that the patient was prevented from becoming any worse, it is proper to point out that this patient was in a stationary phase, and that the treatment may have had nothing to do with his remaining in this condition. If, on the other hand, it could be shown that the patient at *B* showed definite improvement in circulation, the evidence of the value of the treatment would be more convincing. Similarly if the downward course of the patient at *C* were interrupted and reversed, the value of the treatment would be demonstrated.

Table II presents an individual case which illustrates such spontaneous improvement. An occlusion took place in this patient's left calf in November, 1927. Previously the oscillometer reading at the left calf was 4. After the occlusion it dropped to ¼. He was treated from November, 1927, to June, 1928, during which time the oscillometer readings repeatedly taken showed a change from ¼ to ½. At this time, for economic reasons, the patient stopped treatment. Repeated examinations in the four following years nevertheless showed a continued rise in oscillometer readings to 2½. In contrast to the recovery that took place in the left calf the oscillometer readings in the

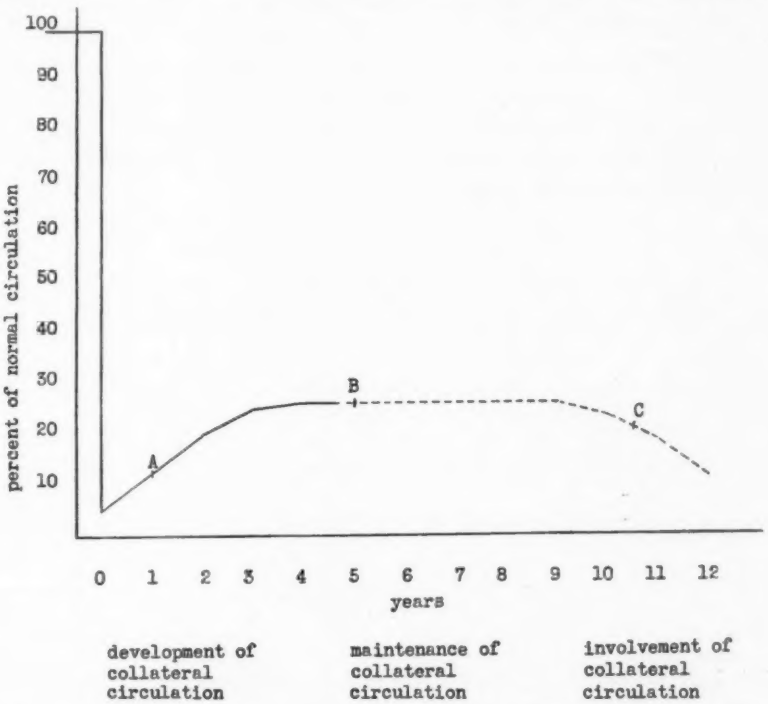

Fig. 1.—Spontaneous improvement in circulation following sudden closure.

right calf in response to treatment, and during the years after treatments were stopped, remained at practically the same level. In the right calf the occlusion had taken place years before in 1924. The right leg was in a stationary phase and it remained in this phase throughout the period of observation.

It has been claimed that prevention of gangrene following occlusion of a major artery demonstrates the value of a method of treatment. If the tendency to spontaneous improvement is borne in mind, it is readily apparent that such evidence in the individual case is of little value. In a recent report from the Mayo Clinic on 100 patients who had had acute occlusion and who had received little treatment of any

kind, it was stated that 50 per cent failed to develop any gangrene.[1] A similar case is the following. A patient, fifty years of age, developed a coronary thrombosis. He remained in bed for four weeks and then began to walk around. Two weeks later the right lower extremity suddenly became numb and cold, obviously due to an embolism in the right femoral artery. Since this patient was of rather stolid type and apparently did not have very severe pain, he did nothing about it for four weeks. I first examined him at this time, and it was evident that the circulation in the major vessels of the right lower extremity had been almost completely occluded four weeks before. The oscillometer readings at the calf and ankle were 0. In spite of this serious impairment of circulation no gangrene had supervened. If this patient had been treated following the acute closure by the suction-pressure apparatus or any other form of treatment, it would no doubt have been claimed that the treatment was responsible for the preservation of the extremity. As a matter of fact spontaneous recovery was sufficient to avoid gangrene.

TABLE II

OSCILLOMETER READINGS ILLUSTRATING SPONTANEOUS IMPROVEMENT

ACUTE CLOSURE IN NOVEMBER, 1927 LEFT CALF			ACUTE CLOSURE IN 1924 RIGHT CALF
October, 1927	4		1
November, 1927	¾		1
December, 1927	¾		1
February, 1928	¾	Period of treatment	1
May, 1928	½		1
June, 1928	½		1
August, 1928	½		½
June, 1929	1¼		½
March, 1930	2	No treatment	¾
February, 1931	2½		1

A third form of improvement which is often cited as demonstrating the value of treatment is the healing of ulcers. Anyone who has had considerable experience in dealing with cases of peripheral vascular disease, soon learns that most ulcers tend to heal spontaneously. In an occasional case in which an ulcer has been present for years, and has shown no tendency to heal in spite of rest in bed, satisfactory healing under a method of treatment is impressive evidence of the value of such treatment. In general, however, one should be cautious in accepting such evidence.

These various phases of spontaneous improvement must be borne in mind and evaluated before conceding that a method of therapy has demonstrated value in the treatment of peripheral vascular disease.

The rôle of tobacco in the question of improvement must also be evaluated. It is well known that the smoking of two or three cigarets

results in marked vasoconstriction of the peripheral vessels.[2, 3, 4] This has been shown both by plethysmographic and by skin temperature studies.[2, 3, 4]

Figure 2 shows vasoconstriction after smoking, by a determination of the temperature of the fingers. During the smoking of two cigarets the temperature of the fingers fell from 32° C. to 26° C., a drop of 6 degrees. After cessation of smoking there was a gradual return to a normal temperature. This effect of smoking is present in individuals with normal circulation as well as those with peripheral vascular disease. The constant use of tobacco undoubtedly maintains the vessels in a greater degree of vasoconstriction than would be normal. There may be other ways in which the constant use of tobacco affects ad-

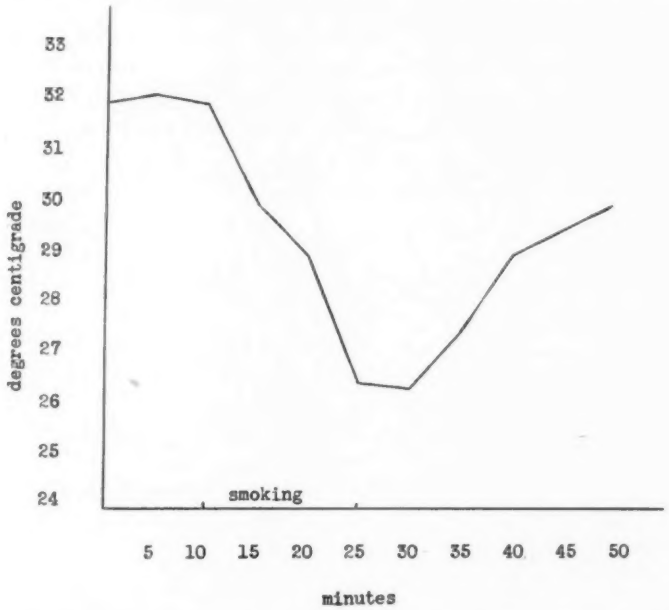

Fig. 2.—Fall in temperature of fingertips while smoking two cigarets.

versely the circulation in the extremities. Clinical experience has amply demonstrated that the cessation of the use of tobacco in itself results in a definite improvement of circulation. In 20 instances of thrombo-angiitis obliterans in my experience where the disease was in a relatively early stage, cessation of smoking without any other form of therapy resulted in improvement or disappearance of all symptoms.

Table III shows the changes in such a case. The patient was a physician forty years of age suffering from intermittent claudication. He had an oscillometer reading of 1½ at the ankle of the affected leg, which still indicated a fair circulation. I felt that active treatment was not necessary and advised merely cessation of smoking. A grad-

ual increase will be noted in oscillometer readings at the ankle from April, 1929, to May, 1930. Associated with this increase there was complete disappearance of symptoms. In May, 1930, apparently not convinced of the relationship between the use of tobacco and his symptoms, this patient resumed smoking. When seen in November the oscillometer reading had been reduced to 1 and there had been a return of symptoms. The patient again stopped smoking at this time and has not used tobacco since. He has now been followed up to May, 1937. The gradual improvement in circulation is evident. This patient is now entirely symptom-free and is engaged in an active practice which requires considerable activity. He had no treatment whatsoever.

Many similar instances could be cited. It is, therefore, apparent that if a patient is induced to stop smoking at the same time that a method of treatment is begun, the improvement that results may be entirely due to cessation of the use of tobacco and not at all to the treatment. In order to be certain that the cessation of the use of tobacco plays no rôle in improvement, smoking must have been stopped at least six months before treatment was started.

TABLE III

OSCILLOMETER READINGS ILLUSTRATING IMPROVEMENT AFTER CESSATION OF SMOKING WITHOUT OTHER TREATMENT

	LEFT ANKLE	
April, 1929	1½	
October, 1929	2½	
May, 1930	3	
		Resumed smoking
November, 1930	1	
		Stopped smoking
February, 1931	2½	
June, 1931	3	
December, 1931	3	
June, 1932	3	
January, 1933	3	
April, 1933	3	
April, 1934	3½	
April, 1935	3½	
May, 1936	3½	
May, 1937	4½	

Finally it is necessary to consider the relationship of vasoconstriction to this question of evaluation of treatment. Until recent years there were no satisfactory objective methods of measuring circulation in the legs. Two methods that have been developed are now in general use. One of these is measurement by means of the oscillometer, and the second, measurement of skin temperature by means of electric thermometers. Increases in oscillometer readings and in the temperature of the toes are now frequently cited as objective evidence that the method of treatment employed has increased the circulation

in the extremities. While such objective evidence is valuable and convincing, there are certain errors in interpretation that must be avoided.

Regulation of the body temperature is a vitally important function. It is an extraordinary fact that changes in environmental temperature of as much as 50° F. are promptly compensated for by the regulatory mechanisms of the body so that the internal temperature remains unchanged at 98.6° F. One of the most important of these mechanisms is the regulation of the amount of blood flow in the extremities by means of vasoconstriction. To conserve heat when the environmental temperature is low the peripheral vessels are constricted to reduce the flow of blood. To increase heat elimination these vessels are dilated. The surface temperature at the tips of the extremities is usually much less than the forehead temperature, and the difference may be as much as 15° Centigrade.

Changes in the size of the peripheral vessels due to vasoconstriction are reflected in both oscillometer measurements and surface tempera-

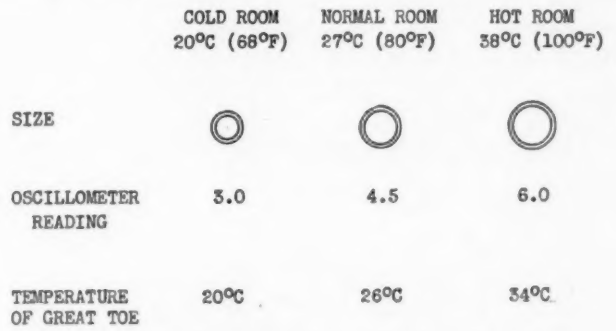

Fig. 3.—Physiologic variations in normal blood vessels.

ture readings. Thus in a cold room with the vessels constricted the oscillometer reading at the ankle which is normally between 4 and 5 may be reduced to 3 or less (Fig. 3). In a hot room the reading may be as much as 6. Similarly the surface temperature of the great toe may be 20° C. in a cold room and 34° C. in a hot room. It is obvious that these fluctuations in oscillometer measurements and surface temperature readings do not indicate any disease in the blood vessels. They are normal variations which are characteristic of all healthy human beings.

The degree of vasoconstriction in peripheral vessels is similarly influenced by the mental state of the patient. Increased nervousness or emotional disturbance is likely to produce constriction, while mental relaxation will reduce this tendency. Such changes, also, are characteristic of all normal people.

Since the function of body heat regulation by means of vasoconstriction is so vitally important for good health, it is not surprising

that nature does not relinquish it when there is disease of the peripheral arteries. Even with advanced organic disease of the peripheral vessels there is still some superimposed vasoconstriction reducing the amount of blood flow to the extremity. It is only in the last stages of peripheral vascular disease, when the integrity of the extremity is actually threatened by loss of blood supply, that nature reluctantly gives up this ability to vary the volume of peripheral circulation.

Figure 4 illustrates a vessel whose lumen has been encroached upon by organic disease. When vasodilatation is produced in such a vessel by environmental changes the lumen is increased in size and more blood can flow through it.

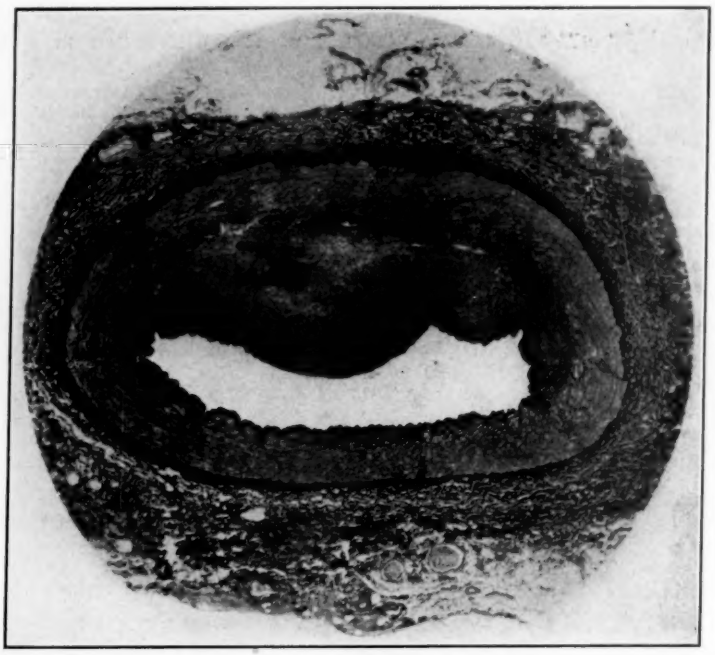

Fig. 4.—Cross-section of vessel showing encroachment of lumen by disease process.

Such alterations in the size of this diseased vessel will be reflected in variations of oscillometer measurements and surface temperature readings. These changes are diagrammatically represented in Fig. 5. When increased oscillometer measurements and surface temperature readings are offered as objective evidence of improvement in circulation, one must be sure that they are not normal variations due to differences in environmental temperature or in the patient's mental state.

How then is one to avoid this difficulty? There are two factors responsible for the reduction in blood flow. One is the actual reduction of the lumen of the blood vessels by organic disease. The second is the superimposed vasoconstriction. If all vasoconstriction could be abolished temporarily, the reduction in blood flow would then be due

only to the organic disease present. Measurements with the oscillometer and the skin thermometer under such conditions would reveal the real degree of improvement resulting from treatment.

There are many methods of temporarily eliminating vasoconstriction. For example any form of anesthesia accomplishes this purpose.

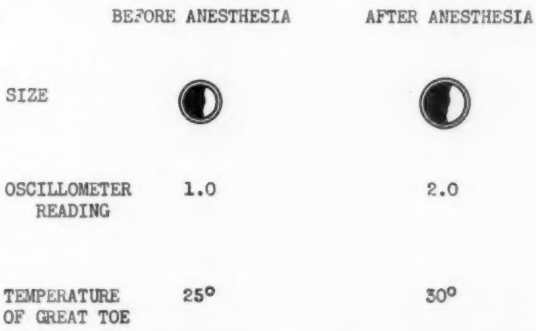

Fig. 5.—Diagram to illustrate changes in size of lumen of diseased vessel.

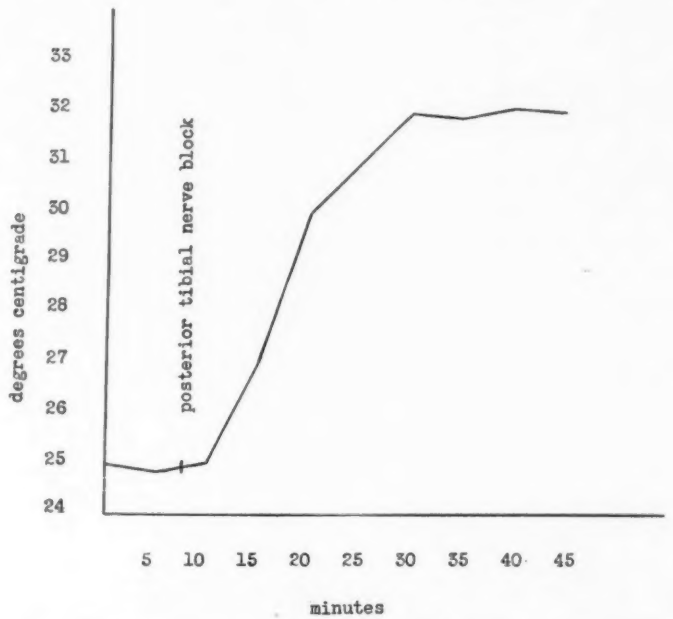

Fig. 6.—Changes in temperature of great toe following release of vasoconstriction by means of anesthesia.

General or spinal anesthesia may be employed, paravertebral injections of novocaine, or novocaine injections of the peripheral nerves. It has been found by experience that no matter which form of anesthesia is used, relatively complete vasodilatation is produced.[5] For practical purposes in clinic or office practice, the simplest form of anesthesia is most suitable.

This is accomplished by a novocaine injection of the posterior tibial nerve at the ankle. This procedure releases vasoconstriction in the vessels of the foot. The increased blood flow causes an elevation of temperature which attains its maximum in about fifteen or twenty minutes (Fig. 6). The oscillometer measurement taken at the ankle reflects the maximum dilatation possible in the diseased vessels. The maximum temperature of the great toe and the maximum oscillometer measurements obtained under such conditions are objective measurements of the circulation when the vasoconstrictor factor is completely eliminated. A comparison can be made under similar conditions six months later, and if at that time increased readings can be demonstrated, the evidence of improvement is convincing. Table IV illustrates comparative readings made in this manner.

TABLE IV

COMPARATIVE MEASUREMENTS OF CIRCULATION BEFORE AND AFTER TREATMENT

	BEFORE ANESTHESIA	AFTER ANESTHESIA
	Before Treatment	
Oscillometer reading	1.0	1.5
Temperature of toe	25	29.5
	After Treatment	
Oscillometer reading	1.5	2.5
Temperature of toe	25	31.5

All of the cases coming for treatment to a large circulatory clinic are not suitable for evaluating the results of treatment. From among them, however, certain patients can be selected who may serve this purpose. The ideal case is an individual who has had intermittent claudication for at least two or three years so that the phase of spontaneous improvement has passed; one who has stopped smoking for more than a year; and perhaps one who has been treated for a considerable period by some method without improvement. This will rule out any psychic factor due to contact with the physician and will show that spontaneous improvement is not taking place. The circulation in such a patient should be studied by means of oscillometer readings and temperature studies after complete vasodilatation has been produced under anesthesia. He is now ready to be treated. If during the next year he shows steady subjective improvement and this improvement is supported by objective evidences, such as increased oscillometer and temperature readings under controlled conditions, we may permit ourselves to accept such evidence as indicating the value of the treatment.

SUMMARY

During the past decade a great variety of methods of treatment have been proposed to improve the circulation in individuals with peripheral vascular disease. Critical evaluation of these methods requires that

certain factors be determined. These are the tendency to spontaneous improvement after arterial occlusion, the effect of cessation of smoking and the normal variations in vasoconstriction due to environmental changes in temperature and in the patient's psychic state. Evidence of improvement can be accepted as indicating the value of a form of treatment only if it is shown that these factors have been eliminated.

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PAROXYSMAL BUNDLE-BRANCH BLOCK ASSOCIATED WITH HEART DISEASE

A Review and an Analysis of the Literature, With Thirteen New Cases and Notes Upon the Influence of the Vagus*

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THE increasing number of cases of transient or recurrent bundle-branch block which have been reported during the past few years indicates that this condition is not an uncommon one. Many of these cases have been classified as essentially functional, a term which in many instances is misleading. From a study of the literature and from our own material it is evident that a clear differentiation is important between the group associated with organic heart disease and the group described by Wolff, Parkinson, and White[57] with apparently congenitally wide QRS waves and short P-R intervals. In the latter, the delay in intraventricular conduction is of no serious significance. In the former the periods of bundle-branch block are primarily due to abnormal changes in the conducting tissue and are to be considered a manifestation of advanced heart disease. In some cases of this group certain physiological factors, particularly changes in vagal tone, may indirectly influence intraventricular conduction and be immediately responsible for the appearance or disappearance of branch block. In such cases, however, it is not justifiable to conclude that the vagal action is primarily or wholly responsible for the fluctuations in intraventricular conduction.

That type of bundle-branch block which is associated with very short P-R intervals has been thoroughly defined and well investigated and will be described only briefly in this report. The cases to be reported are confined to the group associated with organic heart disease with the possible exception of one case (Case 1). There are thirteen examples of this type of intermittent or transient bundle-branch block which have come to our attention during the past few years. In five cases we have recorded the transition from one degree of conduction to another. Observations directed toward studying the effect of changes in vagal tone in causing transitions to or from bundle-branch block were made in six cases.

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Cases With Wide QRS Waves and Short P-R Intervals

This group belongs to the type of bundle-branch block studied and described by Wolff, Parkinson, and White in 1930.[57] They presented a syndrome which is characterized by a distinctive electrocardiographic pattern, consisting of wide QRS complexes with short P-R intervals, occurring usually in young persons without other evidence of heart disease who are prone to attacks of paroxysmal auricular tachycardia, flutter, or fibrillation. There may be a reversion to the normal form spontaneously, during the paroxysmal tachycardia, after exercise or after the administration of atropine or quinidine. Holzmann and Scherf[23] in 1932 and Wolferth and Wood[56] in 1933 have published critical studies of this condition and have independently suggested a very plausible explanation of its mechanism. They believe that the abnormal ventricular complex does not represent a block in a bundle branch but that it is due to the early arrival of an auricular impulse in one ventricle by way of a short-circuiting conducting bundle, such as a bundle of Kent. Although this is the probable mechanism, the electrocardiographic abnormality is still commonly classified as bundle-branch block and it will be so termed in this report. Some of these cases have now been followed for a number of years without manifestation of heart disease and consequently this condition is best considered at the present time simply as an unusual variation of the normal cardiac mechanism.

There should also be included among the inconsequential cases those rare individuals with no apparent cardiovascular disease in whom a bundle-branch block manifests itself after a prolonged period of paroxysmal tachycardia. This is interpreted as indicating fatigue of one of the bundle branches by the prolonged rapid rate. It has been pointed out, however, that this is most likely to occur in individuals in whom there is heart disease, so that such a finding should probably always be regarded with some suspicion.

TYPE ASSOCIATED WITH HEART DISEASE

In selecting cases from the literature, as with our own cases, we have limited ourselves to the classical form of bundle-branch block with Q-S intervals of 0.12 second or over, except for a few instances in which the intraventricular conduction time was over 0.10 but under 0.12 second. In the latter a marked change to or from a bundle-branch block shape of QRS complex in association with the prolongation of intraventricular conduction seemed sufficient to warrant their being interpreted as branch block. We have not included cases in which there has been only a slight change in the intraventricular conduction time or in which only isolated complexes showed bundle-branch block. Throughout this report the nomenclature proposed by Wilson and his coworkers[4, 55] will be employed to indicate the bundle branch which is affected.

CASES

Cases 1 to 8 have been observed by us. Cases 9, 10, 11, and 12 were taken from hospital records. Case 13 was seen in private practice by an associate.* In the following cases the pulse and heart rates have been measured in beats per minute, the blood pressure in millimeters of mercury systolic and diastolic, and the Q-S and P-R intervals in seconds, and will be recorded simply numerically in the reports.

CASE 1.—A. S., female, aged forty-one years, entered the Boston City Hospital on June 21, 1935, and was discharged on August 31, 1935.

She complained of the progressive development of severe pain and swelling of her ankles for four days. For the preceding five weeks she had had vague migratory joint pains. There had been no recent infection and there were no cardiovascular symptoms. The past history was negative for rheumatic fever, arthritis and chorea.

Examination showed a patient who, although not acutely ill, was in obvious pain. The temperature was 100.2° F. and the pulse rate was 108. A soft, localized, apical, systolic murmur was the only abnormal physical finding in the heart. X-ray films of the heart showed it to be normal in size and shape. The blood pressure was 110/70. Both feet and ankles were swollen and exquisitely tender on motion. With the exception of a few carious teeth no other abnormalities were found.

Laboratory Data.—Repeated urine examinations, red and white blood cell counts, and hemoglobin determinations were normal. The corrected sedimentation rate (Ernstene method) showed the high value of 1.2 mm. per minute when done a few days after admission. Periodic determinations showed a gradual fall to 0.40 mm. per minute during the course of the following eight weeks. The blood Kahn reaction was negative for syphilis. Cervical smears revealed no gonococci and the gonococcal complement fixation test was reported as doubtful. The ankle joints were normal by x-ray.

Course.—She became clinically well after a few days. The temperature and joint pains subsided under sodium salicylate therapy. The systolic murmur disappeared and there was no clinical evidence of heart disease. The patient was followed regularly through 1936 and remained well, with no definite signs or symptoms of heart disease.

Electrocardiograms.—(Fig. 1.) During the eight weeks in the hospital eight routine electrocardiograms were taken and all but two showed right bundle-branch block (Q-S 0.12-0.14, P-R 0.16-0.18). The normal records (Q-S 0.07-0.08, P-R 0.18-0.20) appeared in the fourth and sixth weeks. Records showing right bundle-branch block were also obtained in September and October, 1935, and April, November, and December, 1936. The cardiac rate during branch block varied between 60 and 125, while during normal conduction the rates were 70 and 71. All records showed rather low amplitude of the QRS complexes but there were no T-wave changes in the records showing normal conduction.

Diagnosis.—Acute rheumatic fever.

Observations.—Observations were made on August 13, 14, 15, 16, and 30, 1935. Bundle-branch block was present when each of the following procedures was applied. No opportunity was presented for studying their effects on normal conduction because the observed normal periods were too short.

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*For permission to include Cases 8 and 9, Case 10, Case 11, and Case 13, we are indebted respectively to Dr. Cadis Phipps, and Dr. J. A. Foley of the Boston City Hospital, and Dr. Ashton Graybiel and Dr. H. B. Sprague of the Massachusetts General Hospital. For an electrocardiogram in Case 7 we thank Dr. C. R. Comstock of Saratoga Springs, N. Y.

Carotid Sinus Pressure: On August 13 transitions to normal were associated with carotid sinus pressure on five out of nine trials. On all of these occasions the transitions to normal conduction accompanied a reduction in heart rate and with reversion to a higher rate bundle-branch block reappeared. These transitions were usually associated with gradual alterations in heart rate although the changes in conduction took place suddenly without intermediate complexes. During the periods of normal conduction the heart rate varied between 75 and 85 while during bundle-branch block it ranged from 79 to 94. The rates below 85 with bundle-branch block occurred in those cycles just prior to transitions to normal conduction. On the occasions when no transitions occurred with carotid sinus pressure the minimum rate recorded was 85.

The effects of carotid sinus pressure were tested sixteen times in the course of the subsequent observations in August. No transitions to normal conduction occurred in spite of slowing of the rate which in two records on August 31 reached 49 and 54.

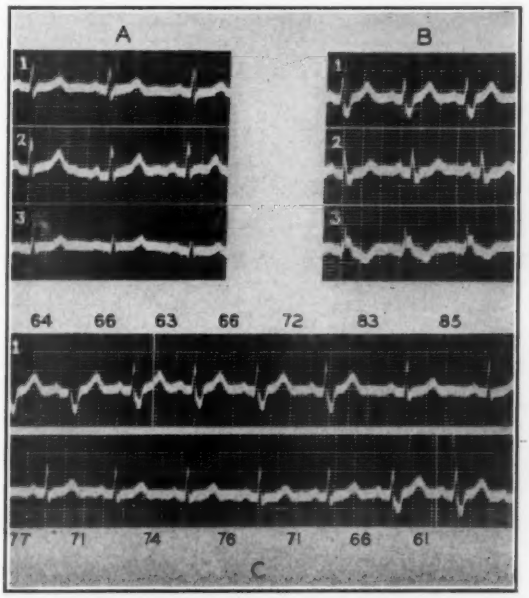

Fig. 1.—Case 1. *A*, (Aug. 5, 1935) normal conduction. *B*, (Sept. 27, 1935) right bundle-branch block. *C*, (Aug. 13, 1935) continuous record showing transition from right bundle-branch block to normal conduction produced by bilateral carotid sinus pressure (duration indicated by the vertical white signal lines) and return to branch block with release of pressure. Numbers refer to the R-R intervals and are in 0.01 sec. Time-marker 0.20 and 0.04 sec.

This slowing was the maximum obtained at any time. In the other fourteen trials the minimum heart rate was 85. Although slowing occurred, similar negative results were obtained in December, 1936.

Oculocardiac Reflex: This was tested on August 13 and 15. On the thirteenth the rate slowed from 94 to 75, with a change to normal complexes appearing when the rate was 79. On the fifteenth there was slowing from 100 to 85 without a transition.

Posture: On changing the position from sitting to reclining or vice versa transitions were recorded on two of six trials. On August 15, bundle-branch block was present at a rate of 85 in the sitting position and normal conduction at a rate of 75 in the reclining position. On August 16, normal conduction was present in both positions at a rate varying from 72 to 85 except for one period of six branch

block complexes (rate 75 to 79) which appeared and disappeared while the patient was sitting. On August 14 and 30, branch block was present in both positions with a minimum rate of 79.

Effect of Mild Exercise: This was tested on August 13 and 14. No records were taken prior to exercise. On the former date the rate slowed from 94 to 88 without improvement in conduction. On the latter the rate slowed from 88 to 79, normal conduction being present when the rate was 85 or less.

Nitroglycerine: Nitroglycerine, 0.65 mg., was administered in the usual manner. No alteration from branch block took place and the rate reached 130.

Atropine: Atropine sulfate, 1.0 mg., was injected intravenously. The heart rate rose to 135 and bundle-branch block persisted throughout.

Spontaneous Transition: On August 14 normal conduction (rate 85 to 88) was present throughout a control strip lasting 17.7 seconds, except for one period of six branch block complexes at a rate of 88.

Comment.—Although it is difficult to draw general conclusions from these observations the factor of cardiac rate seems to be an important one. Except for two short runs of bundle-branch block, normal conduction was always associated with a slower cardiac rate than the preceding or succeeding periods of block. This was particularly well shown on August 13 when transitions occurred at an apparently critical heart rate of about 85. The evidence indicates that the cardiac rate during the observations was a major factor in determining the presence or absence of bundle-branch block in the early period of the patient's course. The fact that it became progressively more difficult to influence the block during the latter part of August 1935 and the fact that normal conduction never appeared subsequently, either spontaneously or when every attempt was made to induce it a year later, strongly suggest that an organic factor was present which eventually caused permanent block to be established.

That the block was not the result of direct vagal influences was demonstrated by the inability of atropine to abolish it. It is quite probable that the increased heart rate which followed the administration of nitroglycerine may have offset any effect which coronary dilation might have had in improving the conduction.

Although we cannot definitely determine the nature of the organic changes which progressively damaged the right bundle branch we are tempted to attribute it to pathological changes incidental to a rheumatic infection. It is possible that rheumatic involvement of the small coronary twig supplying the right bundle branch may have resulted in gradual occlusion and ultimate fibrosis of the branch.* However, there is sufficient reason to warrant caution in definitely classifying the etiology of the branch block as rheumatic since there were no other manifesta-

*Yater has recently reported, at the annual scientific meeting of the American Heart Association (June 8, 1937), six new cases of bundle-branch block studied carefully post mortem by serial sections. Two of these patients had chronic rheumatic heart disease and their electrocardiograms had shown right bundle-branch block. In both, Yater found complete interruption of the right bundle branch with less damage to the left. He suggested that rheumatic involvement of the branches of the coronary artery supplying the conducting tissue may have been the pathological process in these cases.

tions of rheumatic carditis and since it is known incidentally that delayed A-V conduction in acute rheumatic fever usually disappears. At present, however, the rheumatic etiology seems more likely than any other.

CASE 2.—C. McD., female, aged thirty-eight years, entered the Boston City Hospital for the first time Jan. 3, 1936, and was discharged April 16, 1936.

She had been active and well until three months before admission, at which time dyspnea on exertion gradually appeared and became progressively worse. Edema of the feet and ankles had appeared a month before entry. There was no previous history of rheumatic infection.

Examination showed a slightly enlarged heart with a regular rhythm and a rough but faint apical systolic murmur. X-ray cardiac measurements were: to the right of the median line 3.4 cm., to the left 9.9 cm., internal diameter of the thorax 25.0 cm. and great blood vessels 5.5 cm. The blood pressure was 105/65. The lungs were clear. The liver edge was 3 cm. below the costal margin and there was moderate pitting edema up to the knees and in the sacral region. No other abnormalities were observed.

Laboratory Data.—Urine and blood examinations were negative on admission but the leucocyte count rose after ten days in the hospital and remained between 10,000 and 14,000 per cu. mm. until discharge. The corrected sedimentation rate (Ernstene method) fluctuated between 2.90 and 0.70 mm. per minute. The Kahn test on the blood was negative for syphilis. Total serum proteins were 6.2 gm. per cent, with an albumin-globulin ratio of 1.0. Blood cultures showed no growth.

Course.—The patient was digitalized and given routine cardiac treatment. There was temperature up to 100° F. in the evenings and a pulse rate between 90 and 100 for two weeks. She was then given acetylsalicylic acid and the temperature and pulse rate promptly subsided. Symptomatically she improved gradually but she was kept in the hospital for a number of weeks because there was evidence of active infection.

The patient was readmitted on Oct. 16, 1936, and was discharged on Feb. 20, 1937.

Since her previous admission she had remained fairly well except for slight dyspnea on exertion until one month before readmission when she began to experience dull nonradiating substernal pain unrelated to food or exertion. This pain came during night or day and lasted about an hour. The dyspnea on exertion became worse, palpitation was more marked, and edema of the ankles returned.

Examination revealed essentially the same signs in regard to the cardiovascular system except that a slight bulge was noted on fluoroscopy in the region of the left auricle which was interpreted as a rheumatic deformity. Some dullness, diminished breath sounds, and moist râles were found at the right lung base. A varying amount of edema was present during her stay in the hospital.

Laboratory Data.—Urine and blood examinations were negative except for the leucocyte count which varied between 5,000 and 13,500. The corrected sedimentation rate was 0.68 mm. per minute on admission and thereafter fluctuated between 0.40 and 1.30 mm. per minute. The serum proteins were normal.

Course.—For a time she improved, but periodic low fever persisted, and dyspnea and edema recurred when she was allowed up. On Feb. 1, 1937, her tonsils were removed and bilateral tonsillar abscesses drained. Following this operation there was a distinct but gradual improvement and she was discharged nineteen days later, fever, dyspnea, and edema having disappeared.

Electrocardiograms.—(Fig. 2.) From January to April, 1936, nine routine electrocardiograms were taken. The first two, taken on January 9 and 16, showed left bundle-branch block (Q-S 0.12-0.14, P-R 0.20). The remaining seven showed

normal conduction (Q-S 0.07-0.08, P-R 0.16-0.20). During the second admission ten routine records were taken. The first, on October 17, showed left bundle-branch block (Q-S 0.12-0.14, P-R 0.16). On December 14 there was delayed A-V conduction (Q-S 0.08, P-R 0.23) and on February 10, 1937, intraventricular conduction was slightly prolonged (Q-S 0.10-0.11). The remaining seven records taken at intervals throughout her course showed normal conduction (Q-S 0.07-0.08, P-R 0.16-0.20). The records showing normal intraventricular conduction also showed left axis deviation and diphasic or inverted T-waves in Lead I, attributed to digitalis. In neither admission was there a constant correlation between the cardiac rate and the form of the electrocardiogram.

Diagnosis.—Myocarditis, ? rheumatic.

Observations.—On October 19 an observation was made on the influence of respiration. Three transitions from normal conduction (Q-S 0.06, P-R 0.14) to left bundle-branch block (Q-S 0.10-0.12, P-R 0.14-0.16) occurred spontaneously during

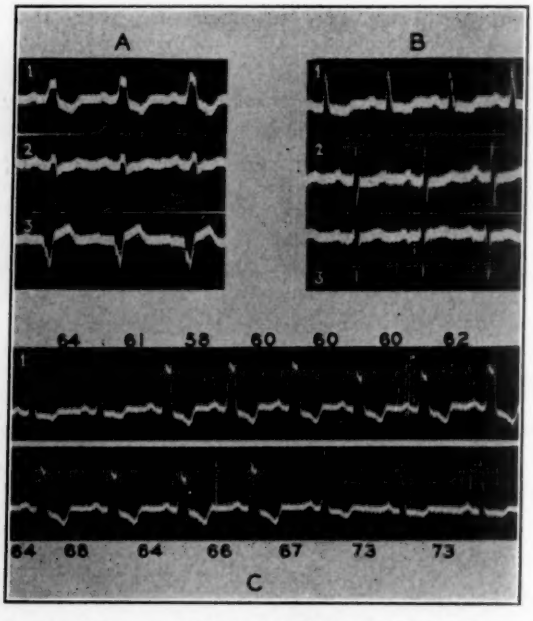

Fig. 2.—Case 2. *A*, (Jan. 9, 1936) left bundle-branch block. *B*, (Feb. 8, 1936) normal condition. *C*, (Oct. 20, 1936) continuous record showing spontaneous appearance and disappearance of left bundle-branch block during quiet respiration in the sitting position. Numbers refer to the R-R intervals and are in 0.01 sec. Time-marker 0.20 and 0.04 sec.

quiet respiration in the recumbent position. Deep sustained inspiration and expiration did not change normal conduction to branch block. On October 20 no periods of branch block were observed during quiet respiration, inspiration, expiration, or carotid sinus pressure with the patient recumbent. In the sitting position, however, periods of left bundle-branch block (Q-S 0.11-0.12, P-R 0.14) were recorded on nine occasions, the longest lasting eighteen seconds. Although the effects of the procedures mentioned above were tested, these periods of defective conduction all occurred during quiet respiration either before or after some procedure was carried out. On October 21 one short period of bundle-branch block (9 complexes) was recorded during quiet respiration in the recumbent position. In the sitting position, however, during quiet respiration consecutive records by the three standard leads covering fifty-nine seconds showed branch block throughout.

It was an invariable finding during these observations that when bundle-branch block was present the heart rate was greater than during normal intraventricular conduction. The periods of branch block showed rates varying from 86 to 104 while the rates during normal conduction varied between 62 and 95. The average difference in rate between periods of defective conduction and the preceding or succeeding periods of normal conduction was 10 beats per minute, with extremes of 4 and 16. The changes in rate were gradual and occasional transitional complexes were seen. It should be noted that here, as in Case 1, although there was an association between the cardiac rate and the form of the electrocardiogram at the time of these observations, no such association existed from day to day as manifested in the routine tracings.

Comment.—In each admission left bundle-branch block was recorded during the early period when the clinical evidence of poor myocardial function was most marked. With improvement the branch block disappeared. Consequently we feel that the tendency toward block was dependent primarily upon the state of the myocardium and that the cardiac rate, at times, was the immediate factor determining the absence or presence of defective conduction. The natural tendency for the heart rate to increase when a person rises to the sitting position may account for the fact that transitions to branch block occurred more frequently in this position.

CASE 3.—M. R., negress, aged fifty-one years, was admitted to the Boston City Hospital April 14, 1936, and was discharged May 30, 1936.

She complained of having had slowly increasing dyspnea on exertion for five years and of swelling of the ankles for one year. During the four months prior to admission she had become much worse, with dyspnea at rest and attacks of paroxysmal nocturnal dyspnea. There was no substernal pain. She had known of arterial hypertension for three years before entry.

Examination revealed great dyspnea and slight cyanosis. There was marked narrowing of the retinal arteries and the peripheral arteries were thickened and tortuous. The heart was enlarged, the apex impulse being in the fifth intercostal space 3 cm. beyond the midclavicular line. X-ray cardiac measurements were: to the right of the median line 6.0 cm., to the left 11.0 cm., internal diameter of the thorax 25.0 cm., and great blood vessels 8.0 cm. The heart showed general enlargement. The rhythm was regular, but the heart sounds were of poor quality. There was a protodiastolic gallop rhythm and a high pitched blowing apical systolic murmur. The blood pressure was 168/140. Numerous moist râles were heard at the lung bases. The liver edge was not felt and there was no dependent edema.

Laboratory Data.—The urine showed an occasional trace of albumin. The urine concentration test showed a minimum specific gravity of 1.008 and a maximum of 1.012. The phenolsulphonephthalein test showed an excretion of 55 per cent of the dye in two hours. The blood nonprotein nitrogen varied between 26 and 30 mg. per cent. The total blood serum protein was 5.1 gm. per cent.

Course.—With routine cardiac therapy the patient made a slow recovery but her convalescence was interrupted by bronchopneumonia. The blood pressure fell to 140/100. She was followed after her discharge and when last seen on April 15, 1937, she complained of substernal oppression, dyspnea on exertion, palpitation, and nervousness. There was no edema. Her blood pressure was 204/130.

Electrocardiograms.—(Fig. 3*A*.) During her hospital stay six routine records were taken. On May 7 and 27 there was normal conduction (Q-S 0.10, P-R 0.16-

0.18) with rates of 68 and 70 respectively. The records on April 15, May 14, 19, and 20 showed left bundle-branch block (Q-S 0.16, P-R 0.18) with rates varying from 67 to 85. During eleven months following discharge eight records were obtained. In June, July, and November, 1936, and January and April, 1937, left bundle-branch block (Q-S 0.14-0.16, P-R 0.14-0.16) was present with rates varying from 85 to 105. In August, 1936, and February and March, 1937, there was normal conduction (Q-S 0.10, P-R 0.16) with rates of 78 to 81. When normal conduction was present the records showed left axis deviation with inverted T-waves in Lead I and low upright or inverted T-waves in Lead II.

Diagnosis.—Hypertensive heart disease with left ventricular failure. Bronchopneumonia.

Observations.—On May 19, 0.3 c.c. of amyl nitrite was inhaled. The heart rate rose from 109 to 124. No alteration to normal conduction took place. On May 25 normal intraventricular conduction was present throughout in spite of deep sustained inspiration and expiration. On Jan. 7, 1937, when bundle branch block was present the application of right and left carotid sinus pressure did not alter either the heart rate or the conduction time. When normal conduction was present on February 4 carotid sinus pressure again failed to influence either the rate or the conduction and sustained inspiration and expiration were likewise ineffective. Climbing one flight of stairs raised the rate from 77 to 108 but normal conduction remained.

Comment.—No correlation was found between the clinical state of the patient and the presence or absence of branch block. It is possible that the cardiac rate played some rôle in determining the degree of intraventricular conduction, but the T-wave changes strongly suggest that coronary artery disease affecting the left bundle branch was the major factor in this patient.

CASE 4.—P. D., male, aged fifty-six years, was admitted to the Boston City Hospital for the first time May 27, 1936, and was discharged June 4, 1936.

He complained of weakness of the right side of the face and inability to speak both of sudden onset and of one day's duration.

Examination revealed a right facial paralysis of the central type and a motor aphasia. The heart was enlarged. X-ray cardiac measurements were: to the right of the median line 4.5 cm., to the left 10.0 cm., internal diameter of the thorax 29.0 cm. and great blood vessels 6.5 cm. The rhythm was normal but there was a loud rough apical systolic murmur. The blood pressure was 220/118. There were many moist râles at both lung bases. No dependent edema was present.

Laboratory Data.—The urine contained a trace of albumin and the blood nonprotein nitrogen was 28 mg. per cent. Blood cell counts were normal. The blood Hinton reaction was negative for syphilis.

Course.—The patient improved gradually and was discharged with a slight residual paralysis and moderately slurred speech. The blood pressure on discharge was 185/110.

He was readmitted to the hospital Oct. 4, 1936, and was discharged Oct. 17, 1936.

At this time he complained of increasing dyspnea and fatigue for three weeks. He had had several attacks of paroxysmal nocturnal dyspnea during this period. A recent respiratory infection had increased his symptoms.

Examination revealed essentially the same clinical and x-ray findings as before. There was still a residual right facial weakness and slurred speech. The blood pressure was 235/120.

Laboratory Data.—Urine and blood were essentially normal. The phenolsulphonephthalein test showed 65 per cent excretion of the dye in two hours. The urine concentration test gave a minimum specific gravity of 1.008 and a maximum of 1.011.

Course.—He was digitalized and with routine cardiac care he improved rapidly. The blood pressure on discharge was 185/95. He was seen again in January, 1937. He had considerably restricted his mode of living and as a result he had few cardiovascular symptoms. At this time slight apical systolic and faint aortic diastolic murmurs were found. The blood pressure was 180/76.

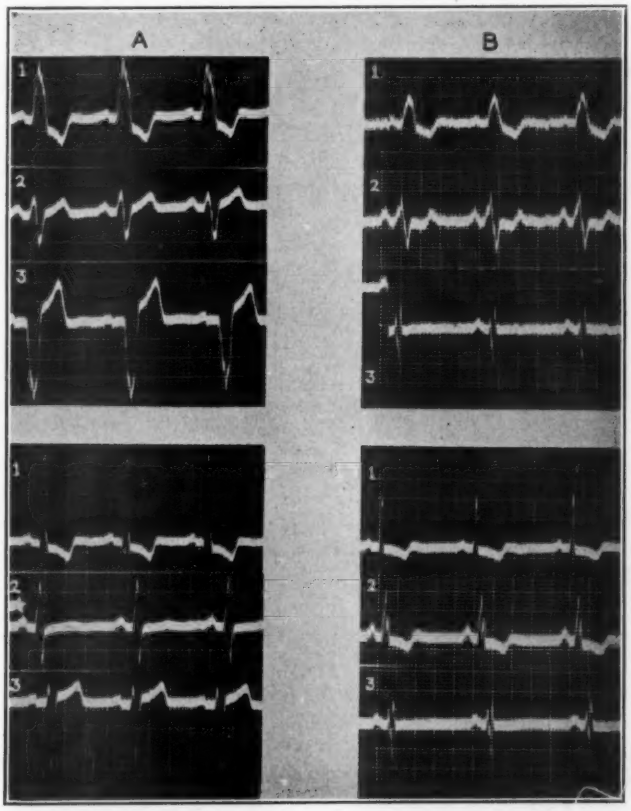

Fig. 3.—*A*, Case 3, upper, (May 20, 1936) left bundle-branch block. Lower, (May 27, 1936) normal conduction.
B, Case 4, upper, (Oct. 6, 1936) left bundle-branch block in Leads I and II, normal conduction in Lead III.
Lower, (Oct. 8, 1936) normal conduction. Time-marker 0.20 and 0.04 sec.

Electrocardiograms.—(Fig. 3*B*.) The only record taken during his first admission showed normal conduction (Q-S 0.10, P-R 0.14) with a rate of 70. Two days after readmission the first routine record showed left bundle-branch block (Q-S 0.16, P-R 0.16) in Leads I and II, with a rate of 67, while Lead III showed normal conduction (Q-S 0.08, P-R 0.16), with a rate of 62. Three more routine records at this time and two taken in January, 1937, all showed normal conduction (Q-S 0.08-0.10, P-R 0.16-0.20) at rates varying from 60 to 75. In all records the T-waves in Leads I and II were diphasic or inverted and usually were of low origin. Left axis deviation was present.

Diagnosis.—Hypertensive and coronary heart disease with slight aortic regurgitation. Left ventricular failure. Cerebral thrombosis.

Observations.—On October 9 sustained inspiration and expiration, as well as changing the posture from lying to sitting, did not alter normal conduction. Right carotid sinus pressure with the patient recumbent induced sinus standstill and an idioventricular rhythm at a rate of 35. The same procedure in the sitting position induced sino-auricular bradycardia (rate 48). Left carotid sinus pressure in both positions induced complete A-V dissociation (auricular rates 46 to 48, ventricular 40 to 48). No change to bundle-branch block occurred. On January 11, 1937, right and left carotid sinus pressure induced sinus standstill with an idioventricular rhythm (Q-S 0.08-0.10) at rates of 33 to 37.

Comment.—The only recorded episode of bundle-branch block in this patient occurred during a period of left ventricular failure and it is probably to be related to the temporary myocardial dysfunction and possibly to coronary artery disease. The block was probably intermittent during this period in spite of the fact that only one such episode was recorded.

CASE 5.—L. N., male, aged forty years, was admitted to the Boston City Hospital for the first time on Jan. 19, 1926, and was discharged Feb. 6, 1926.

He gave a previous history of severe acute rheumatism at the ages of fourteen and thirty-six years. Three weeks before entry he caught a "cold" and migratory joint pains developed four days before he came to the hospital. For five years he had noticed palpitation and some dyspnea on exertion but he had not been incapacitated by these symptoms.

Examination revealed a slightly enlarged heart, with the left border of dullness 10.5 cm. to the left of the midsternal line. The heart rhythm was regular and the rate was 65. A blowing apical systolic murmur and an early blowing diastolic murmur at the base were present. The blood pressure was 125/85. Moist râles were heard at the lung bases but engorgement of the cervical veins, palpable liver and edema were absent.

Laboratory Data.—The urine and blood examinations and Wassermann reaction were negative.

Course.—He was treated with sodium salicylate and his joint symptoms cleared rapidly.

He entered the hospital for the second time Dec. 19, 1932, and was discharged Jan. 24, 1933.

For two weeks prior to admission he had suffered from frequent "fainting spells." His cardiovascular symptoms had remained of mild degree.

Examination showed the heart to be of essentially the same size as before. The rhythm was regular. (Variations in cardiac rates are given with electrocardiograms.) A systolic thrill was felt in the aortic area and there were present characteristic aortic and mitral systolic and diastolic murmurs. The blood pressure was 122/70. There was no evidence of heart failure.

Course.—While in the hospital he had frequent syncopal attacks when no heart beats were heard. He was treated with ephedrine sulfate by mouth. These seizures ceased after two weeks.

At the age of fifty years he was admitted for the third time Dec. 30, 1936, and was discharged Jan. 7, 1937.

He had been followed since his previous discharge and in 1934 his pulse had become permanently slowed at a rate of approximately 35. He had, however, experienced

no syncopal attacks until the day of his admission when he had two while in bed. Slight dyspnea on exertion had persisted but there were no other cardiovascular symptoms.

Examination showed no essential changes from the findings recorded above except for a heart rate of 38 and a blood pressure of 135/70. X-ray cardiac measurements were: to the right of the median line 6.0 cm., to the left 11.0 cm., internal diameter of the thorax 29.0 cm., and great blood vessels 5.5 cm. There was no evidence of heart failure.

Course.—He remained comfortable and had no syncopal attacks. Ephedrine sulfate was given without appreciable change in heart rate.

He was admitted for the fourth time April 1, 1937, and he died April 6, 1937.

Sore throat and epistaxis developed three weeks before entry and were followed by hot painful swelling of the wrists. The joint pains and malaise persisted until admission. His cardiovascular symptoms had remained unchanged.

Examination showed large injected tonsils and mild cervical adenitis. The wrists were red, swollen, and tender. The cardiovascular findings were essentially the same as were present on the previous admission. The temperature was 101.6° F.

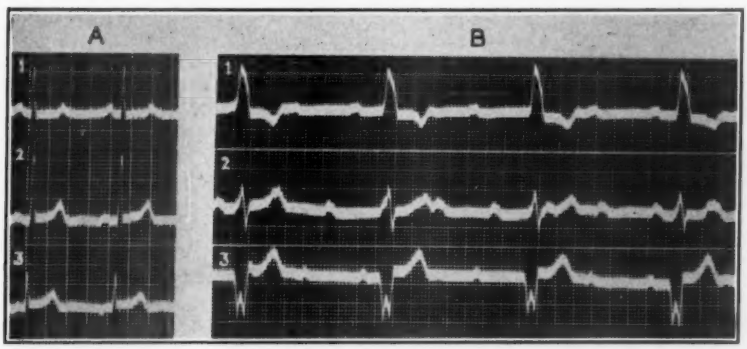

Fig. 4.—Case 5. *A*, (Jan. 21, 1933) normal conduction. *B*, (Jan. 5, 1937) left bundle-branch block and complete A-V dissociation. Time-marker 0.20 and 0.04 sec.

Laboratory Data.—The urine contained a small trace of albumin. The blood was normal.

Course.—He was given sodium salicylate and the fever disappeared in twenty-four hours. The heart rate on admission was 95, but on the evening of the second day he had three attacks characterized by convulsions and loss of consciousness. At this time the cardiac rate was found to be 28. He was given ephedrine and had no more attacks. He appeared to be improving but on the fifth day was found dead in bed.

Electrocardiograms.—(Fig. 4.) During his first admission two routine records showed normal sinus rhythm (Q-S 0.06, P-R 0.18-0.20) with rates of 60 to 67. In the early part of his second admission one record showed complete A-V dissociation with left bundle-branch block (Q-S 0.14), two showed complete A-V dissociation with a lesser degree of intraventricular block (Q-S 0.12-0.14) and one showed 2:1 A-V dissociation with left bundle-branch block (Q-S 0.16, P-R 0.20). The ventricular rate varied from 41 to 46. During the last four weeks of this hospital stay two records showed slight retardation of intraventricular conduction (Q-S 0.11-0.12) and four were normal (Q-S 0.06-0.10, P-R 0.14-0.20) the rate varying from 60 to 84. Two routine records were taken during the third admission. Both showed complete

A-V dissociation and left bundle-branch block (Q-S 0.16) with ventricular rates of 43 and 40. During his last stay in hospital three records were taken, all on April 2. At 10 A.M. there was complete A-V dissociation, the auricular rate being 110 and the ventricular 88, with right bundle-branch block (Q-S 0.14); at 4:30 P.M., after a syncopal seizure, complete A-V dissociation was still present with the same auricular rate but the ventricular rate had fallen to 24 and left bundle-branch block (Q-S 0.16) had reappeared; at 9:30 P.M. complete A-V dissociation was shown and the ventricular complexes were those of left bundle-branch block, but minor variations in the shape of the ventricular complexes at this time indicated a varying site of origin in the junctional tissue. The auricular and ventricular rates were 130 and 26 respectively. No axis deviation or T-wave changes were noted in the normal records.

Autopsy.—The heart weighed 710 gm. The right ventricle was markedly dilated and its wall measured 0.4 cm. in thickness. The left ventricular wall was 2.0 to 2.5 cm. in thickness but the cavity was not dilated. The tricuspid and pulmonary valves were normal. The mitral valve, 6.8 cm. in circumference, was greatly narrowed, the leaflets were thickened, and the chordae tendinae were shortened and thickened. The aortic valve, 6.1 cm. in circumference, showed marked interadherence of the cusps which were thickened, retracted, and fixed. There was no evidence of acute endocarditis. The openings of the coronary arteries were unaffected and the vessels showed minimal atherosclerosis. Apart from chronic passive congestion of the liver, spleen, and kidneys and atelectasis of the lower parts of both lungs there were no other gross pathological findings. Microscopic examination of the heart showed dense fibrous scarring of the aortic ring, the membranous part of the septum, and the upper part of the muscular portion of the septum under the left ventricular endocardium. In addition, throughout the heart, including the upper part of the septum, there were many areas of intense infiltration with lymphocytes, plasma cells, and fibroblasts mostly perivascular in position. There were distinct Aschoff nodules to be seen. The conduction system was not studied.

Diagnosis.—Acute rheumatic fever. Acute and chronic rheumatic heart disease with aortic and mitral stenosis and regurgitation.

Observations.—On January 5, 1937, right and left carotid sinus pressure and forced inspiration and expiration against closed air passages, all in both the sitting and recumbent positions, and the inhalation of 0.17 c.c. of amyl nitrite failed to influence the ventricular rate or intraventricular conduction.

Comment.—In 1933 there was transient, and in 1937 persistent, complete A-V dissociation and left bundle-branch block, the pathological basis for which is to be found in the wide-spread fibrosis in the region of the aortic ring and the upper part of the interventricular septum. The appearance a few days before death of a rapid ventricular rate in the presence of the complete A-V dissociation with complexes of right-branch block form may be attributed to the irritation of a lower impulse center by the acute rheumatic process. This center was probably situated below the lesion in the left branch and thus produced complexes of right branch block type. When the irritation disappeared the former and slower center above the level of the branch block again dominated the ventricles and the left bundle-branch block reappeared. An alternate explanation suggested by Yater[60] is that there was damage to both bundle branches, more advanced on the left, with the usual pacemaker being situated in

the right branch below the lesion on that side. However, for the reasons given above, it is probable that the pacemaker in this case shifted for a short time to the left branch below its lesion and right bundle-branch complexes ensued.

CASE 6.—J. H., male, aged fifty-seven years, was admitted to the Massachusetts General Hospital April 6, 1937, and was discharged April 27, 1937.

He had been perfectly well until three weeks before entry when he began to feel tight, low substernal pain when walking, which disappeared immediately with rest. Two days before admission while at rest he had one attack of pain which radiated down both arms. At 2:30 A.M. on the morning of entry he was awakened by a con-

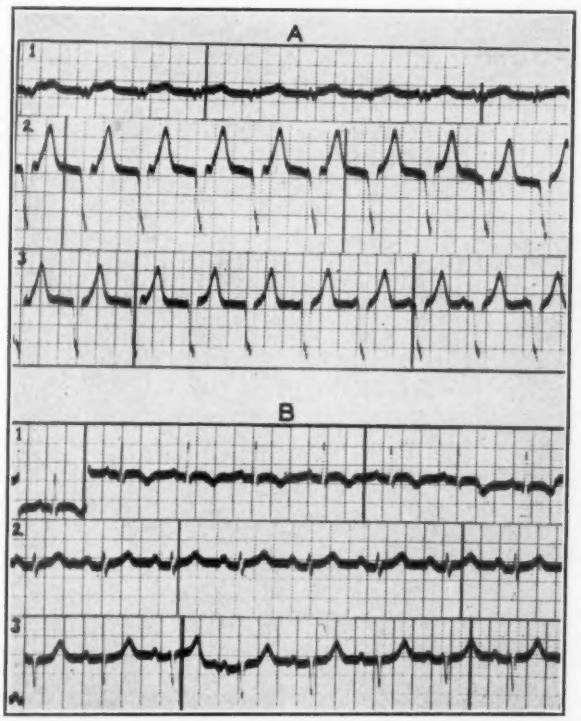

Fig. 5.—Case 6. *A*, (April 6, 1937) left bundle-branch block and complete A-V dissociation. *B*, (April 7, 1937) normal conduction. Time-marker 0.2 and 0.1 sec.

stant, severe, substernal pain which gradually disappeared during the course of the following fifteen hours. There were no other symptoms except some belching of gas and sweating.

Examination eight hours after the onset of pain revealed a man who did not appear particularly ill. The cardiac apex impulse was felt in the fifth intercostal space 9 cm. from the midsternum. The cardiac rhythm was regular and there were no murmurs, pericardial friction, or gallop rhythm. The blood pressure was 142/100. No signs of heart failure were present.

Laboratory Data.—The blood leucocyte count was 14,000 per cubic millimeter on admission. The urine was normal. The blood Wassermann and Hinton reactions were negative for syphilis.

Course.—The temperature on admission was 101° F. During the course of the next five days this gradually fell to a normal level. The blood pressure established itself at a level of 130/80. He was kept in bed and given aminophyllin. His convalescence was uneventful.

Electrocardiograms.—(Fig. 5.) Eight electrocardiograms were taken during the hospital stay. The first, taken eight hours after the onset of pain, showed left bundle-branch block (Q-S 0.13) with what was apparently complete A-V dissociation with a rapid ventricular rate. The auricular and ventricular rates were both 100. On the following day and in the subsequent records the A-V and intraventricular conduction were normal (Q-S 0.07-0.08, P-R 0.20). There was moderate to marked left axis deviation in all records showing normal conduction. The Q-wave in Lead IV was absent in all records and there were progressive changes in the T-waves of Leads I, II, and IV in the last seven records indicating a recent myocardial infarct.

Diagnosis.—Coronary heart disease. Acute coronary thrombosis (anterior type).

Comment.—There can be little doubt that the conduction disturbances observed in this patient were due to either direct or indirect effects of

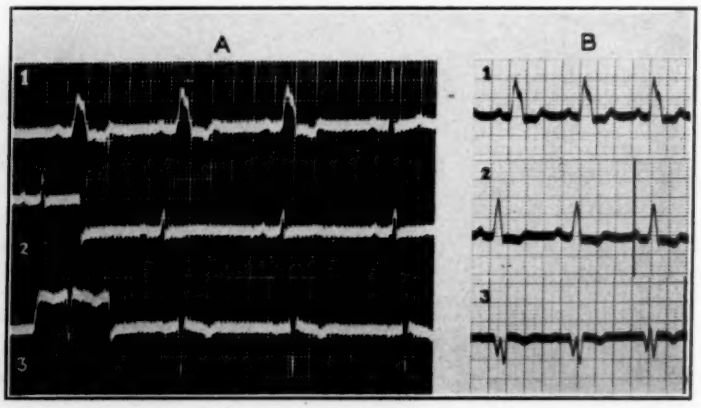

Fig. 6.—Case 7. *A*, (September, 1936) Lead I spontaneous transition from left bundle-branch block to normal conduction; Leads II and III, normal conduction. Time-marker 0.20 and 0.04 sec. *B*, (March 16, 1937) left bundle-branch block. Time-marker 0.2 and 0.1 sec.

a myocardial infarct involving the bundle of His. Temporary interference with the local circulation or involvement of the conduction system by the less irrevocable changes occurring at the periphery of the infarcted area can explain their transient nature.

CASE 7.—T. A., female, aged fifty-four years, was first seen in private consultation at the Massachusetts General Hospital in August, 1936.

At that time she complained that she was awakened at night by severe attacks of substernal pain which radiated into the left arm. For the previous six months she had noticed a tight substernal feeling when walking which occasionally radiated down the left arm. This would subside after five or ten minutes of rest. All of these symptoms had recently been considerably relieved by nitroglycerine and aminophyllin. Arterial hypertension of 170/110 had been discovered on routine examination in 1931.

Examination revealed an enlarged heart. The orthodiagraphic measurements were: transverse diameter of the heart 12.0 cm., internal diameter of the thorax

21.8 cm. The rhythm was regular. The aortic second sound was accentuated and there was a slight basal systolic murmur. The blood pressure was 150/100. No other abnormalities were found.

Electrocardiograms.—(Fig. 6.) Four electrocardiograms were taken. Left bundle-branch block (Q-S 0.16, P-R 0.19) was present in June and August, 1936. In September, 1936, the electrocardiogram showed a transition from branch block (Q-S 0.16, P-R 0.19) to normal complexes (Q-S 0.08, P-R 0.19) in Lead I. The normal type persisted through Leads II and III. The cardiac rate during block ranged from 63 to 59 while during normal conduction the rate varied from 52 to 56. A record taken on March 16, 1937, showed left branch block throughout (Q-S 0.17).

Diagnosis.—Hypertensive and coronary heart disease. Angina pectoris decubitus.

Comment.—Angina pectoris decubitus indicates considerable coronary insufficiency and it is probable that variability of a partially deficient blood supply to the conducting tissue was the primary factor determining the presence or absence of branch block.

CASE 8.—J. R., male, aged forty-one years, was first admitted to the Boston City Hospital Jan. 15, 1935, and was discharged Feb. 7, 1935.

He complained of increasing dyspnea on exertion for one year and of a vague dull precordial pain present when he was tired. No other cardiovascular symptoms were elicited.

Examination was negative except for a blood pressure of 160/110.

Laboratory Data.—Urine and blood were normal.

Course.—He improved with bed rest and symptomatic treatment. The blood pressure on discharge was 125/90.

He was admitted for the second time on June 18, 1936, and was discharged on June 28, 1936.

At this time he complained of increasing dyspnea on exertion for six months, orthopnea for three months and severe attacks of paroxysmal nocturnal dyspnea for one week.

Examination showed slight distension of the cervical veins and an enlarged heart. X-ray cardiac measurements were: to the right of the median line 7.0 cm., to the left 12.5 cm., internal diameter of the thorax 30.0 cm., and great blood vessels 9.5 cm. The rhythm was regular and there were no murmurs. The blood pressure was 140/100. A tender liver edge was felt 3 cm. below the costal margin. Moist râles were present at both lung bases. There was no peripheral edema.

Laboratory Data.—The urine and blood were normal.

Course.—He was digitalized and given diuretics. Under this regime improvement was rapid.

He entered the hospital for the third time July 25, 1936, and was discharged Sept. 15, 1936.

On the day of admission he had a fainting spell. He also complained of increased dyspnea, occasional precordial pain on exertion, and blurring of vision.

Examination revealed essentially the same findings as on his previous admission except that a moderate blowing systolic murmur was heard at the apex. The blood pressure was 138/108. Signs of pulmonary congestion were present but the liver was not felt and there was no peripheral edema.

Course.—While in the hospital he had several moderately severe attacks of paroxysmal nocturnal dyspnea which were relieved by morphia. His digitalis ration was increased, diuretics were administered, and he improved gradually. He was seen

again on January 15, 1937, when he complained of dyspnea on exertion. He exhibited a well-marked pulsus alternans and his blood pressure was 138/88.

Electrocardiograms.—(Fig. 7*A*.) All seven electrocardiograms taken between January, 1935, and August, 1936, showed left bundle-branch block (Q-S 0.14-0.16, P-R 0.16-0.18). Four subsequent records in September, 1936, and January, 1937, showed normal conduction (Q-S 0.08-0.10, P-R 0.16-0.18). During branch block the rate varied from 79 to 108 while during normal conduction it ranged between 79 and 97. The records showing normal conduction also showed left axis deviation and well-marked inversion of the T-waves in Leads I and II with low origin.

Diagnosis.—Hypertensive and coronary heart disease.

Observations.—On January 15, 1937, the effects of deep respiration, forced inspiration, and expiration against closed air passages and carotid sinus pressure were tested. The first two procedures had no effect on rate or conduction. Eight trials of carotid sinus pressure each resulted in S-A slowing without A-V block, the minimum rate recorded being 30. On two occasions when slowing was marked (40 and 30) a single broad complex of bundle-branch block type appeared (Q-S 0.16, P-R 0.18) but otherwise no change in conduction occurred. Climbing one flight of stairs induced dyspnea, raised the heart rate from 71 to 88, but did not alter the normal conduction.

Comment.—In this case there was no correlation between the form of the electrocardiogram and the clinical condition of the patient. The T-wave changes indicate coronary disease and the persistence of normal conduction since September 1936 may possibly be associated with the establishment of collateral circulation and better vascularization of the conducting tissue.

CASE 9.—B. B., female, aged forty-four years, was first admitted to the Boston City Hospital April 6, 1934, and was discharged April 14, 1934.

She complained of increasing dyspnea on exertion for six months. Arterial hypertension had been discovered during a pregnancy in 1928 and had been present since that time, with nervousness and headaches.

Examination showed the heart to be enlarged, with the apex impulse in the fifth intercostal space 12.5 cm. to the left of the midsternum. There was a soft basal systolic murmur. The blood pressure was 270/150. The lungs were clear and no edema was present.

Laboratory data.—The urine had a specific gravity of 1.015 to 1.017 and contained a trace of albumin. The blood nonprotein nitrogen was 34 mg. per cent.

Course.—After rest and sedation she was discharged improved, with a blood pressure of 180/120.

The patient was readmitted Sept. 10, 1935, and she died Nov. 30, 1935.

Because of increasing dyspnea she had been in and out of bed during the interval since discharge. For seven months she had had increasing edema of the lower extremities and frequent attacks of paroxysmal nocturnal dyspnea. She had taken three grains of digitalis daily for the preceding three months.

Examination revealed the cardiac apex to be in the midaxillary line. X-ray cardiac measurements were: to the right of the median line 7.2 cm., to the left 11.7 cm., internal diameter of the thorax 25.3 cm., and great blood vessels 6.5 cm. The rhythm was regular and no murmurs were present. The blood pressure was 270/170. Both lungs were dull on percussion and moist râles were present. Fluoroscopy revealed a slight amount of pleural fluid bilaterally and hilus congestion. There was edema of the legs, abdominal wall, and back. A tender liver edge was felt 5 to 6 cm. below the costal margin.

Laboratory Data.—The urine examinations showed a small to large trace of albumin, with occasional granular casts. The concentration test showed a minimum specific gravity of 1.010 and a maximum of 1.014. The phenolsulphonephthalein test showed only 25 per cent excretion of the dye after two hours. The blood nonprotein nitrogen varied between 35 and 55 mg. per cent. There was a moderate normochromic anemia.

Course.—Under treatment with bed rest, digitalis, diuretics, and other routine cardiac measures the manifestations of heart failure gradually diminished during

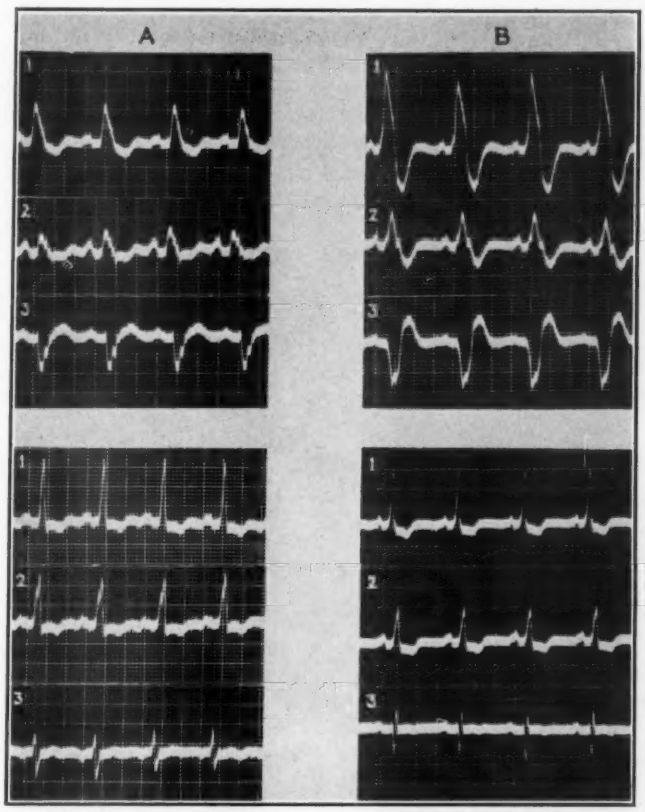

Fig. 7.—*A*, Case 8, upper, (June 19, 1936) left bundle-branch block. Lower, (Sept. 2, 1936) normal conduction.

B, Case 9, upper, (Sept. 11, 1935) left bundle-branch block. Lower, (Sept. 26, 1935) normal conduction. Time-marker 0.20 and 0.04 sec.

the first month. The blood pressure fell to 220/110. Edema, however, returned in the fifth week and ascites and pleural fluid gradually accumulated and persisted in spite of vigorous therapeutic measures. In the eighth week the temperature, previously normal, suddenly rose and signs of infection at the left lung base appeared. From this time onward she failed more rapidly and died in the tenth week.

Electrocardiograms.—(Fig. 7 *B*.) Eleven electrocardiograms were taken, of which six showed normal conduction (Q-S 0.09-0.10, P-R 0.12-0.16) with rates varying from 88 to 107, and five, including one record taken during the early part of the first admission, showed left bundle-branch block (Q-S 0.16-0.20, P-R 0.12-0.14) the rates ranging from 77 to 97. Of the normal electrocardiograms four occurred during the first month of the second admission when improvement was taking place. Left bundle-branch block was present on entry and again during the latter part of her

course when failure recurred. Normal conduction, however, was present on two occasions during this terminal period. There was no constant correlation between the form of the electrocardiogram and the cardiac rate in these routine records. Of the six records with normal conduction all showed inversion of the T-waves in Leads I and II with low origin. The electrical axis was normal.

Autopsy.—(Limited to the heart and kidneys.) The heart weighed 660 gm., with the left ventricle measuring 2.5 cm. in thickness and the right 0.8 cm. The coronary arteries were minimally affected, showing only slight yellow intimal thickening which caused no narrowing. Microscopic examination of the myocardium showed many small scattered fibrous scars with a slight amount of infiltration by polymorphonuclear cells and macrophages. In one section there was a fresh thrombus which almost completely occluded the lumen of an arteriole. The conduction system was not studied. The kidneys together weighed 200 gm. and were of the granular type with the cortex measuring 3 to 5 mm. Microscopic examination revealed many cortical scars with hyalinized glomeruli and atrophied tubules. There was arteriolar narrowing with thrombi in various stages of organization.

Diagnosis.—Hypertensive heart disease with failure. Vascular nephrosclerosis, possibly malignant.

Comment.—The bundle branches were not examined microscopically but it seems likely that the numerous small scars found scattered throughout the myocardium involved the region of the conducting tissue as well and partially damaged a bundle branch. The factor of myocardial failure seems to have been an important one but the fact that normal conduction was present in the latter part of her course indicates an additional factor which may have been circulatory in nature.

CASE 10.—M. S., female, aged forty-five years, was first admitted to the Boston City Hospital Oct. 1, 1936, and was discharged Nov. 12, 1936.

She gave a history of having spent four months in a hospital with rheumatic fever at the age of seven years. Since that time she had had no joint pains but had suffered from frequent sore throats and in recent years had noticed some dyspnea and palpitation on exertion. Six days before entry she had a chill and developed a cough. On the day previous to admission she developed pleuritic pain in the left chest and her ankles, elbows, and shoulders became painful. With the onset of the pulmonary infection increasing dyspnea rendered her unable to sleep.

Examination revealed a cyanotic patient who was markedly dyspneic, with a temperature of 102.5° F. and a respiratory rate of 40 per minute. The cervical veins were engorged. The entire precordium was heaving and the maximum apical impulse was felt in the anterior axillary line. There were a loud systolic murmur and a moderate mid-diastolic rumble at the apex. A basal systolic murmur was also present. The rhythm was regular. The blood pressure was 140/80. Dullness and moist râles were found at both lung bases extending on the left to the angle of the scapula. The abdomen was distended and there was tenderness in the right upper quadrant. Pitting edema was present in the lower extremities. The elbows, shoulders, and especially the ankles were tender.

Laboratory Data.—The urine contained a small trace of albumin. Blood examinations showed a mild normochromic anemia. On admission there was a polymorphonuclear leucocytosis with a count of 21,000 per cubic millimeter which gradually fell to normal. The blood Hinton reaction was negative for syphilis. Two blood cultures produced no growth.

Course.—The patient had a fluctuating fever for three weeks and thereafter with the diminution in the pulmonary signs the fever subsided and the dyspnea improved.

With digitalization the signs of cardiac failure gradually disappeared. The joint pains similarly cleared during the course of the first three weeks. Two weeks before leaving the hospital her pulse which had previously been regular became irregular.

She was readmitted Feb. 21, 1937, and she died March 30, 1937.

In the interval between admissions she was largely confined to bed, complaining of cough, orthopnea, and dyspnea. She was taking three grains of digitalis daily. Three weeks prior to readmission she developed pains in her feet, knees, hands, and elbows and ankle edema appeared.

Examination revealed cervical venous distension. The precordium was heaving with the maximum impulse in the anterior axillary line. X-ray cardiac measurements were: to the right of the median line 5.2 cm., to the left 11.0 cm., internal diameter of the thorax 25.0 cm., and great blood vessels 6.0 cm. Systolic and late diastolic murmurs were heard at the apex and a systolic murmur and a greatly accentuated pulmonary second sound at the base. The blood pressure was 160/65. Dullness and diminished breath sounds were found at both lung bases. There was

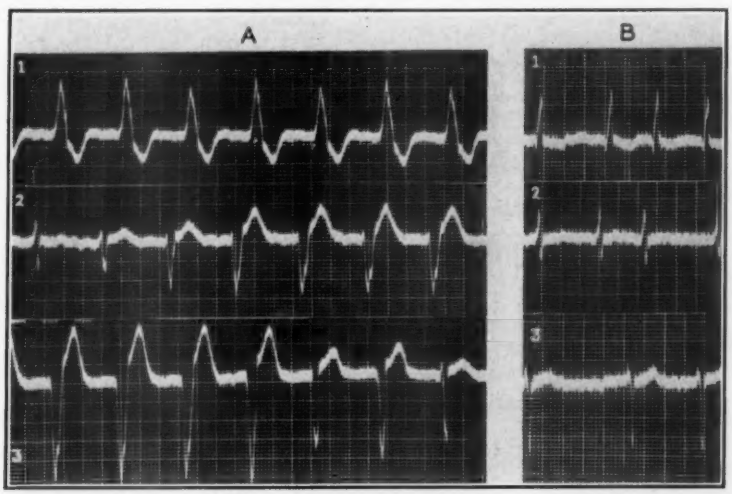

Fig. 8.—Case 10. *A*, (Oct. 5, 1936) auricular fibrillation and idioventricular rhythm. Lead I, left bundle-branch block; Lead II spontaneous transition from normal intraventricular conduction to left bundle-branch block; Lead III reverse transition. Note intermediate complexes. *B*, (Oct. 26, 1936) auricular fibrillation, normal intraventricular conduction. Time-marker 0.20 and 0.04 sec.

slight pitting edema of the ankles. There was stiffness and tenderness of the left ankle and the metacarpophalangeal joints on both sides. The temperature was 99.2° F.

Laboratory Data.—The urine contained a trace of albumin. The blood leucocyte count varied between 7,600 and 11,300 per cubic millimeter. There was a mild hypochromic anemia. The blood nonprotein nitrogen was 24 mg. per cent.

Course.—The slight fever subsided in three days. Local applications relieved the joint pains and with digitalis the cardiac symptoms improved except for occasional bouts of severe dyspnea. On March 20 her throat became sore and joint pains reappeared. She became febrile, cyanosed, and dyspneic. Hemolytic streptococci were grown from throat smears. The temperature rose to 107° F. and the pulse rate to 140 just before death ten days later.

Electrocardiograms.—(Fig. 8.) In the first admission six routine records were taken. On October 2 and 3 there was auricular fibrillation with a regular idio-

ventricular rhythm, the ventricular rates being 80 and 90 respectively and the Q-S intervals 0.08. On October 5 auricular fibrillation with a regular ventricular rhythm at a rate of 88 was still present, but there were, in addition, periods of left bundle-branch block (Q-S 0.14). Several intermediate complexes were recorded at this time. On October 6 the electrocardiogram showed auricular fibrillation and a regular ventricular rhythm at a rate of 81 with left bundle-branch block (Q-S 0.14) throughout. On October 26 and 27 there was auricular fibrillation but the complete irregularity of the ventricular rhythm (rates approximately 80 and 95) indicated resumption of A-V conduction. The intraventricular conduction was now normal (Q-S 0.08-0.10). In the second admission three records were taken. All showed auricular fibrillation and normal intraventricular conduction (Q-S 0.10). The record on Feb. 23, 1937, showed a regular idioventricular rhythm at a rate of 55, while those taken on February 25 and 26 showed an irregular ventricular rhythm at rates of 40 and 45 respectively. Left axis deviation was present in all records.

Autopsy.—The pericardial cavity was obliterated by fibrous adhesions. The heart weighed 530 gm. and showed hypertrophy of the right ventricular wall (thickness 0.8 cm.) and dilatation of the left auricle. The left ventricular wall measured 1.5 cm. in thickness and the cavity was not dilated. There was thickening of the leaflets and thickening and shortening of the chordae tendineae of the mitral valve (circumference 9.0 cm.). The other valves were unaffected. The myocardium showed no abnormality. There was slight atherosclerosis of the coronary arteries. The other gross pathological findings consisted of a chronic adhesive pleuritis on the right side, marked edema of both lungs, and ascites. Microscopic examination showed occasional Aschoff nodules in the myocardium.

Diagnosis.—Acute rheumatic fever. Chronic rheumatic heart disease with mitral stenosis and regurgitation. Congestive failure. Bronchopneumonia.

Comment.—During the first admission, although the patient had a recurrence of acute rheumatic fever, the conduction defects were present at the time when she was seriously ill with chronic rheumatic heart disease and cardiac failure, precipitated by pneumonia. Both the idioventricular rhythm and the bundle-branch block disappeared with clinical improvement. During the second admission although heart failure was present in varying degrees, bundle-branch block was not recorded, but no electrocardiograms were taken during the most severe period.

CASE 11.—H. W., male, aged sixty-five years, was admitted to the Massachusetts General Hospital Sept. 28, 1935, and discharged Nov. 13, 1935.

He had been in good health until the day before admission when he suddenly developed a severe precordial and substernal pain which was only partially relieved by a hypodermic injection of morphine. This pain continued with lessening intensity for the succeeding thirty-six hours. Other than slight dizziness and a desire to eructate there were no associated symptoms.

Examination revealed a moderately sick man, with slight cyanosis of the lips. The cervical veins were not engorged. The heart was slightly enlarged the left border of dullness being in the fifth intercostal space 10 cm. from the midsternal line. The cardiac rhythm was regular and the rate 65. The heart sounds were faint. No murmurs were present. The blood pressure was 120/78. There were a few moist râles at both lung bases but neither enlargement of the liver nor peripheral edema was present.

Course.—On admission the temperature was normal but the blood leucocyte count was elevated to 21,800. Treatment consisted of bed rest, aminophyllin, and symp-

tomatic therapy. In the second week there appeared at the left lung base increased moist râles with bronchial breathing and x-ray opacity. These signs were accompanied by a slight fever and a rise in the blood leucocyte count and were regarded as being due either to bronchopneumonia or to pulmonary infarction. He recovered rapidly from this pulmonary process and had an uneventful convalescence.

Electrocardiograms.—(Fig. 9.) Thirteen routine electrocardiograms were taken during the hospital period. Left bundle-branch block (Q-S 0.15-0.17) was present on September 30 and normal intraventricular conduction (Q-S 0.07-0.09) on the remaining twelve occasions. On September 28, 29, 30, and October 2 there was complete A-V dissociation (auricular rates 80 to 100, ventricular 50 to 65). This was followed on October 3 and 4 by delayed A-V conduction (P-R 0.29 and 0.25). The succeeding records showed normal conduction. Progressive changes from coved S-T segments to late inversion of the T-waves in Leads II and III were observed. In January, 1937, the T-waves in Leads II and III were low while the T-waves in Lead III still showed late inversion. There was moderate left axis deviation in all records.

Diagnosis.—Coronary heart disease. Acute coronary thrombosis (posterior type).

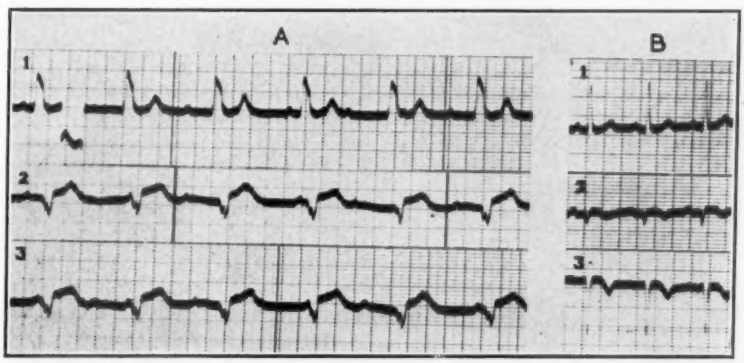

Fig. 9.—Case 11. *A*, (Sept. 30, 1935) left bundle-branch block and complete A-V dissociation. *B*, (Oct. 29, 1935) normal conduction. Time-marker 0.2 and 0.1 sec.

Comment.—In this instance, as with Case 6, the transient conduction disturbances can be correlated with the direct or indirect effects of an acute myocardial infarct.

CASE 12.—H. C., female, aged fifty-five years, came to the out-patient department of the Massachusetts General Hospital Dec. 27, 1935, complaining of dyspnea on exertion and of orthopnea.

Examination revealed a woman somewhat dyspneic at rest. The heart was enlarged with the left border of dullness 13 cm. from the midsternum. The cardiac rhythm was regular. The heart sounds were distant and there was a moderate blowing apical systolic murmur. The blood pressure was 200/100. The breath sounds at the left lung base were diminished and there were a few moist râles at both bases. A tender liver edge was felt 3 to 4 cm. below the costal margin. No edema was present.

Course.—On digitalis and bed rest the patient made considerable improvement during the following two weeks. Her blood pressure, however, remained high (230/130). She was not seen again until March 27, 1936, and at this time she was essentially in the same condition as when first seen. She had been overactive during

the interval and when advised to limit her activities she again improved and a month later there were no manifestations of myocardial failure except dyspnea on moderate exertion. Her blood pressure continued to be elevated. Attempts to follow her failed and she was not seen after May 22, 1936.

Electrocardiogram.—(Fig. 10.) A routine electrocardiogram taken on April 7, 1936, showed left bundle-branch block (Q-S 0.17, P-R 0.17) in Lead I while in Leads II an III there was normal conduction (Q-S 0.06-0.09, P-R 0.17). The cardiac rate was 86 throughout.

Diagnosis.—Hypertensive heart disease with failure.

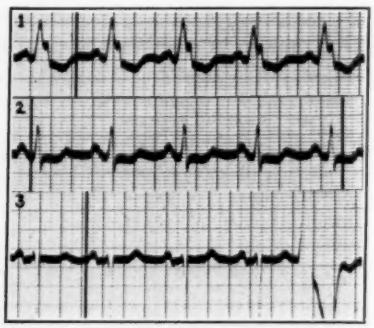

Fig. 10.—Case 12. (April 7, 1936) Lead I, left bundle-branch block; Leads II, and III, normal conduction. Time-marker 0.2 and 0.1 sec.

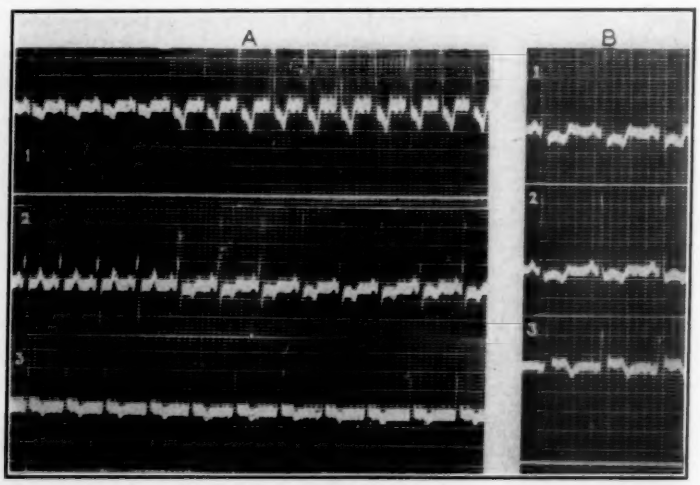

Fig. 11.—Case 13. *A*, (Nov. 12, 1929) Lead I, transition from normal conduction to left bundle-branch block; Lead II, reverse transition; Lead III, normal conduction. *B*, (Aug. 5, 1931) normal conduction. Time-marker 0.1 sec.

Comment.—The only electrocardiogram of this patient was taken during cardiac failure. As in Case 4 the branch block was probably intermittent during this time although only one period of block was recorded.

Case 13.—E. K., female, aged seventy-three years, was first seen in private consultation Nov. 12, 1929, and was followed until death in October, 1931.

She complained of pains across the chest radiating down both arms occurring frequently on exposure to cold or on walking. The pain was relieved by rest. There were no other cardiovascular symptoms.

Examination showed the heart to be slightly enlarged, with the apex impulse in the fifth intercostal space 3 cm. to the left of the midclavicular line. There was a presystolic gallop rhythm. Moderate apical and aortic systolic murmurs were present. The blood pressure was 130/95. No other abnormalities were noted.

Course.—The patient obtained great relief from nitroglycerine and erythrol tetranitrate and a restricted mode of living. The physical findings remained essentially unchanged. The blood pressure was maintained at 150/100. Pulsus alternans developed in November, 1930, and persisted.

Electrocardiograms.—(Fig. 11.) Four routine electrocardiograms were taken. On November 12, 1929, a transition from normal conduction (Q-S 0.08, P-R 0.15) to left bundle-branch block (Q-S 0.12-0.16, P-R 0.15) was recorded in Lead I and the reverse transition occurred in Lead II, after which normal conduction persisted through Lead III. The rate during block ranged from 86 to 92 while during normal conduction it varied between 75 and 80. In December, 1929, normal conduction was recorded in all leads. In May, 1930, there was slight delay in intraventricular conduction (Q-S 0.10-0.11). On August 5, 1931, normal conduction was present in all three leads. There was moderate left axis deviation with diphasic T-waves in Lead I and a coronary type of inversion of the T-waves in Leads II and III, with slightly high origin of the latter in the tracings not distorted by branch block.

Diagnosis.—Hypertensive and coronary heart disease. Angina pectoris.

Comment.—In this instance the clinical and electrocardiographic evidence indicates that insufficiency of the coronary blood supply to the conducting tissue was responsible for the branch block.

In addition to the thirteen cases of paroxysmal bundle-branch block presented above we have collected from the literature fifty-eight additional cases, not of the Wolff, Parkinson, and White type, making a total of seventy-one.* They have been tabulated with the available data in Table I. Sixty-five of these seventy-one cases present clear evidence of heart disease. In Case 1 of our group and in five others (Cases 16, 30, 49, 50, 51) the classical features of cardiac disease were absent and on that account we have excluded them from the following analysis although we believe that the bundle-branch block was in itself evidence of heart disease. Of the remaining sixty-five patients there were thirty-five males, twenty-nine females, and one case in which the sex was not recorded. Thirty-six were over fifty years of age, twenty-six were fifty years of age or under, and in three the age was not recorded. Coronary or hypertensive heart disease was present in forty-four, thirty-three of whom were over fifty years of age; chronic rheumatic heart disease was present in six whose ages ranged from thirty-two to fifty years. The etiology of the heart disease in the remainder was diphtheria, four; thyrotoxicosis, three; congenital, one; and obscure in seven. Left bundle-branch block was present in sixty of the sixty-five cases. It is of interest that there were no instances of right bundle-branch block among the rheumatic cases. In twenty-five patients moderate to severe cardiac failure was associated with the period of block.

*The case reported by Faulkner[15] as due to a rheumatic infection has not been included because subsequent records indicate that it is of the Wolff, Parkinson, and White type.

SUMMARY OF CASES

NUMBER	AUTHOR Series Present	AGE	SEX	DIAGNOSIS	BLOCK TRANSIENT	BLOCK RECURRENT	TRANSITION RECORDED BY E.C.G.	BRANCH AFFECTED	VAGAL STIMULATION EFFECTIVE	TIME BETWEEN ONSET OF B.B.B.* AND DATE WHEN PATIENT WAS LAST SEEN		NOTES
										LIVING	DEAD	
1	Case 1	41	F	Acute rheumatic fever		+	+	Rt.	+	18 mo.		C.S.P. slowed rate and transformed B.B.B. to normal. Later B.B.B. persistent.
2	Case 2	38	F	Myocarditis, ? rheumatic		+	+	Lt.	-	13 mo.		B.B.B. associated with cardiac failure. During controlled observations B.B.B. associated with faster rates. C.S.P. did not induce B.B.B.
3	Case 3	51	F	Hypertensive heart disease		+		Lt.	-	12 mo.		C.S.P. failed to change B.B.B. to normal or vice versa.
4	Case 4	56	M	Hypertensive heart disease	+			Lt.	-	4 mo.		B.B.B. associated with cardiac failure. C.S.P. had no effect on normal conduction.
5	Case 5	50	M	Chronic rheumatic heart disease		+		Lt.	-		52 mo.	B.B.B. associated with transient complete A-V dissociation. Both later became persistent. **Autopsy: Fibrosis in upper part of septum. Death from acute rheumatic carditis.**
6	Case 6	57	M	Coronary thrombosis	+			Lt.			2 mo.	B.B.B. and complete A-V dissociation disappeared in 24 hours.
7	Case 7	54	F	Hypertensive and coronary heart disease		+	+	Lt.		10 mo.		B.B.B. not associated with clinical condition.

*B.B.B.—Bundle-branch block. C.S.P.—Carotid sinus pressure.

SUMMARY OF CASES—CONT'D

8	Case 8	41	M	Hypertensive and coronary heart disease	+			Lt.	-	24 mo.		B.B.B. for 19 mo., normal conduction for next 5. B.B.B. not associated with clinical condition. C.S.P. had no effect on normal conduction.
9	Case 9	44	F	Hypertensive heart disease		+		Lt.			20 mo.	**Autopsy: Myocardial scarring.**
10	Case 10	45	F	Chronic rheumatic heart disease		+	+	Lt.			6 mo.	B.B.B. associated with transient idioventricular rhythm and cardiac failure. Auricular fibrillation also present. **Autopsy: Chronic rheumatic heart disease.**
11	Case 11	65	M	Coronary thrombosis	+			Lt.		1½ mo.		B.B.B., complete, and partial A-V dissociation associated with coronary thrombosis (posterior type). Normal rhythm and conduction resumed.
12	Case 12	55	F	Hypertensive heart disease	+			Lt.		2 mo.		B.B.B. associated with cardiac failure.
13	Case 13	75	F	Hypertensive and coronary heart disease	+		+	Lt.			23 mo.	
14	Azpitarte[1]	62	M	Coronary heart disease	+			Lt.				B.B.B. observed on one day, absent on the following. Associated with probable coronary thrombosis.
15	Bagnaresi[2]	58	M	Hypertensive and coronary heart disease; ? coronary thrombosis		+	+	Lt.	+	18 mo.		C.S.P. and ocular pressure slowed heart and abolished B.B.B. Bellafolina (slowed heart) and lacarnol abolished B.B.B. Amyl nitrite ineffective (rate increased).
16	Baker[3]	75	M	Transient auricular fibrillation		+	+	Lt.			42 mo.	Normal at slow rates, B.B.B. at rapid rates. Oxygen inhalation abolished B.B.B.
17	Boas[6]	63	F	Calcareous aortic stenosis; angina pectoris		+	+	Lt.				Conduction probably dependent on state of coronary circulation.

SUMMARY OF CASES—CONT'D

NUMBER	AUTHOR	AGE	SEX	DIAGNOSIS	BLOCK TRANSIENT	BLOCK RECURRENT	TRANSITION RECORDED BY E.C.G.	BRANCH AFFECTED	VAGAL STIMULATION EFFECTIVE	TIME BETWEEN ONSET OF B.B.B.* AND DATE WHEN PATIENT WAS LAST SEEN		NOTES
										LIVING	DEAD	
18	Bousfield[7]		M	Aortic regurgitation, ? etiology; angina pectoris	+			Lt.				B.B.B. associated with attack of angina pectoris.
19	Campbell[8]	48	F	Chronic rheumatic heart disease		+		Lt.				B.B.B. associated on three occasions with cardiac failure, disappeared on improvement.
20	Campbell and Suzman[9]	52	M	Hypertensive and coronary heart disease		+		Lt.			42 mo.	B.B.B. associated with cardiac failure on two occasions, later became persistent.
21	Caravati[10]	38	M	Coronary thrombosis		+		Lt.		6 mo.		B.B.B. persistent during last 3 mo.
22	Carr[11]	52	M	Coronary heart disease; ? coronary thrombosis		+	+	Lt.	+		44 mo.	C.S.P. and deep inspiration slowed heart and abolished B.B.B.
23	Cowan and Ritchie[12]	43	M	Chronic rheumatic heart disease	+			Lt.				B.B.B. associated with cardiac failure and rapid heart rate. E.C.G.'s in Hill's report.[22]
24	Dressler[15] Fig. 48	57	F	Coronary heart disease	+			Lt.				B.B.B. associated with cardiac failure.
25	Fig. 157	48	M	Coronary heart disease		+		Lt.				Exercise and increase in heart rate induced B.B.B.
26	Fig. 159	50	F	Hypertensive heart disease		+		Lt.				B.B.B. associated with cardiac failure. Periods of 2:1 B.B.B.

*B.B.B.—Bundle-branch block. C.S.P.—Carotid sinus pressure.

SUMMARY OF CASES—CONT'D

27	Elliot and Nuzum[16]	66	M	Coronary heart disease		+	+	Lt.	+	48 mo.		Partial A-V dissociation. Normal complexes after dropped beats. C.S.P. slowed heart and abolished B.B.B. Exercise did not induce B.B.B. Later complete A-V dissociation.
	Freundlich[19]											
28	Case 1	67	F	Hypertensive heart disease		+	+	Lt.	+			C.S.P. slowed heart and abolished B.B.B. In Case 1, B.B.B. associated with cardiac failure. (Two similar cases mentioned without details.)
29	Case 2	63	F	Coronary heart disease		+	+	Lt.	+			
30	Herrmann and Ashman[21] Case 1	55	F	Obscure		+	+	Lt.	+		30 mo.	Indirect vagal action (deep inspiration) slowed heart and abolished B.B.B. Exercise induced B.B.B. In Case 2, B.B.B. associated with cardiac failure.
31	Case 2	47	F	Hypertensive and coronary heart disease		+	+	Lt.	+	26 mo.		
32	Case 3	37	F	Chronic rheumatic heart disease; acute endocarditis; pregnancy		+	+	Lt.		7 mo.		Normal conduction associated with rest.
33	Case 4	60	M	Coronary heart disease		+	+	Lt.				Disappearance of B.B.B. seemed to be associated with clinical improvement.
34	Case 5	65	M	Coronary heart disease		+	+	Lt.				B.B.B. associated with cardiac failure.
35	Hill[22]	52	F	Thyrotoxicosis		+		Lt.				B.B.B. disappeared 2 mo. after thyroidectomy and reappeared 1 mo. later. Auricular fibrillation present until 1 mo. after operation.
36	Hubert[24]	38	M	Diphtheria	+			Lt.				B.B.B. present for 6 mo.
37	von Kapff[25]	45	M	Hypertensive heart disease	+			Lt.				B.B.B. associated with cardiac failure.

SUMMARY OF CASES—CONT'D

NUMBER	AUTHOR	AGE	SEX	DIAGNOSIS	BLOCK TRANSIENT	BLOCK RECURRENT	TRANSITION RECORDED BY E.C.G.	BRANCH AFFECTED	VAGAL STIMULATION EFFECTIVE	TIME BETWEEN ONSET OF B.B.B.* AND DATE WHEN PATIENT WAS LAST SEEN		NOTES
										LIVING	DEAD	
38	Kelly[26]	61	F	Hypertensive heart disease		+		Lt.				2:1 B.B.B. associated with cardiac failure. Complete and persistent B.B.B. appeared after a few days.
	Kurtz[27]											
39	Case 1	73	M	Coronary heart disease	+			Lt.		5 mo.		No cardiac symptoms.
40	Case 2	61	F	Coronary heart disease; coronary thrombosis	+			Lt.		3 mo.		Associated with cardiac failure on two occasions.
41	Case 3	66	M	Hypertensive heart disease		+		Lt.		10 mo.		
42	Case 4	65	M	Coronary heart disease		+	+	Rt.			24 mo.	Associated with cardiac failure.
43	Case 5	56	F	Hypertensive heart disease		+		Lt.				B.B.B. not associated with change in clinical condition. (Insufficient evidence presented for diagnosis of rheumatic heart disease.)
44	Case 6	54	F	Hypertensive and coronary heart disease		+	+	Lt.				B.B.B. associated with cardiac failure. Transient complete and partial A-V dissociation in addition.

*B.B.B.—Bundle-branch block. C.S.P.—Carotid sinus pressure.

SUMMARY OF CASES—CONT'D

45	Lamb[28]	27	F	Thyrotoxicosis		+	0	Lt.		66 mo.		B.B.B. disappeared after subtotal thyroidectomy. Reappeared 19 mo. later as 2:1 B.B.B. during recrudescence of toxicity.
46	Leinbach and White[29]	65	M	Hypertensive and coronary heart disease; angina pectoris		+		Lt.			3-4 yr.	2:1 B.B.B. Later complete and persistent B.B.B.
47	Levine[30] Case 55	57	M	Coronary thrombosis	+			Rt.			15 mo.	Cardiac failure present. B.B.B. disappeared with clinical improvement.
48	Lewis[31]	32	M	Chronic rheumatic heart disease; acute rheumatic fever	+			Lt.				B.B.B. present on one day, absent on the following.
49	von Mentzingen[33]	58	M	"Coronary spasm"		+		Lt.				B.B.B. induced by exercise. Amyl nitrite abolished B.B.B. for 3 minutes.
50	Morris and McGuire[34]	46	F	Acute pulmonary edema		+		Lt.		48 mo.		B.B.B. at first associated with acute pulmonary edema; later became persistent.
51		53	F	Cirrhosis of liver	+			Lt.			60 mo.	B.B.B. appeared during shock following laparotomy; died from syphilitic cirrhosis of liver.
52	Pezzi[35]	49	M	Coronary heart disease		+		Lt.		12 mo.		At times all complexes showed B.B.B., at others B.B.B. intermingled with normal complexes.
53	Robinson[36] Case 3	33	M	Auricular fibrillation		+		Lt.			2 days	B.B.B. associated with cardiac failure and rapid rate.
54	Case 4	59	M	Complete A-V dissociation	+			Lt.				B.B.B. associated with cardiac failure.

SUMMARY OF CASES—CONT'D

NUMBER	AUTHOR	AGE	SEX	DIAGNOSIS	BLOCK TRANSIENT	BLOCK RECURRENT	TRANSITION RECORDED BY E.C.G.	BRANCH AFFECTED	VAGAL STIMULATION EFFECTIVE	TIME BETWEEN ONSET OF B.B.B.* AND DATE WHEN PATIENT WAS LAST SEEN		NOTES
										LIVING	DEAD	
55	Robinson[37]	63	M	Coronary heart disease	+			Lt.				B.B.B. associated with auricular fibrillation and cardiac failure. Heart rate 135. Digitalis slowed rate and B.B.B. disappeared. Six weeks later rate 158 but I-V conduction normal.
56	Rossi[38]			Coronary heart disease		+	+	Lt.	+			C.S.P. and amyl nitrite abolished B.B.B.
57	Slater[41]	40	F	Thyrotoxicosis		+		Lt.				"Myocardial involvement." 3:1 and 4:1 B.B.B. becoming complete and persistent after a few hours.
	Stecher[43]											
58	Case 1	5	M	Diphtheria	+			Lt.			1 mo.	
59	Case 3	13	F	Diphtheria	+			Lt.				B.B.B. disappeared within two weeks.
60	Case 5	7	M	Diphtheria	+			Lt.				B.B.B. disappeared within eight days.
61	Steidl[44]	65	F	Hypertensive and coronary heart disease		+		Lt.		66 mo.		
62	Stenstrom[46] Case 3	50	F	Transient auricular fibrillation		+	+	Lt.				Associated with cardiac failure. B.B.B. dependent on rate. Induced by exercise, abolished by rest.

SUMMARY OF CASES—CONT'D

63	Stenstrom[47] Case 1		M	Coronary and hypertensive heart disease		+	+	Lt.	+	7 mo.		C.S.P. slowed the heart and changed B.B.B. to normal. Later persistent B.B.B. with no effect produced by C.S.P.
64	Case 2	42	F	Hypertensive heart disease		+	+	Lt.	+	48 mo.		C.S.P. slowed the heart and changed B.B.B. to normal. Exercise transformed normal QRS to B.B.B. 2:1 B.B.B. was present at times.
65	Case 3	70	F	Coronary and hypertensive heart disease		+	+	Rt.	-		3 mo.	One tracing (Fig. 23) suggests 2:1 R.B.B.B. B.B.B. associated with heart failure.
66	Willius and Anderson[51]	63	M	Hypertensive heart disease		+	+	Lt.		39 mo.		Delayed A-V conduction with B.B.B.
	Willius and Keith[52]											
67	Case 1	65	F	Hypertensive and coronary heart disease		+		Lt.		60 mo.		B.B.B. later became persistent.
68	Case 2	66	F	Coronary heart disease; ? coronary thrombosis		+		Lt.		14 mo.		B.B.B. associated with cardiac failure.
69	Case 3	46	M	Hypertensive and coronary heart disease	+			Lt.			18 mo.	B.B.B. associated with cardiac failure.
70	Wood, Jeffers, and Wolferth[58, 59]	19	M	Congenital heart disease		+	+	Rt.		36 mo.		Interventricular septal defect. Transition to normal conduction for 3 cycles.
71	[redacted]	55	M	Auricular fibrillation	+			Rt.				

The finding of permanent bundle-branch block is often considered to be of grave prognostic significance. Recent reports,[17, 48, 49, 58] however, indicate that this conduction disturbance per se does not necessarily have the unfavorable influence on prognosis often attributed to it. An attempt was made to follow up the reported cases of paroxysmal bundle-branch block but insufficient data were available to warrant drawing definite conclusions. In general, however, the prognosis in paroxysmal as in permanent bundle-branch block seems to be dependent upon the type and severity of the associated heart disease.

DISCUSSION

Association of Paroxysmal Bundle-Branch Block With Coronary and Hypertensive Heart Disease.—This is the largest group. The organic basis of the block in these cases is probably either arteriosclerotic narrowing in the coronary vessels supplying the conduction tissue, with or without partial permanent damage to a bundle branch,* or the direct or indirect effects of an acute coronary thrombosis. Changes in the conducting fibers accompanying failure of the myocardium may be the primary or an additional factor in the production of the branch block.

The above statements, although at present impossible of absolute proof, are based upon more than hypothetical grounds. It is known that high degrees of A-V dissociation, sometimes transient in character, are observed in cases in which careful histopathological investigation of the conduction system shows partial or no damage. Of some significance are the clinicopathological findings in three cases by Géraudel[20] in which he claimed that permanent and transitory A-V dissociation could be associated with arteriosclerotic narrowing of the artery supplying the conducting tissue, without recognizable damage to the bundle of His. It is not absolutely clear, however, that Géraudel examined the bundle branches as carefully as the main bundle so that bilateral bundle-branch lesions which could have produced the complete A-V dissociation may have been overlooked. Of more significance is the finding of transient increases in intraventricular conduction time,[54] and in one case (Case 18), transient bundle-branch block itself, associated with attacks of angina pectoris. In accordance with the current concepts of angina pectoris these electrocardiographic changes must be attributed to anoxemia of the conducting tissue. Oxygen inhalation (Case 16) and amyl nitrite (Cases 49, 56) have transformed bundle-branch block to normal conduction. Further, the association of bundle-branch block with the clinical evidence of severe heart failure and the return to normal intraventricular conduction with better myocardial function has been observed frequently.

*Yater[60] believes that there is partial fibrosis of one or both bundle branches in all cases of intermittent block.

Thus, although the *precipitating* factor of the bundle-branch block may be physiological in some cases, the *fundamental cause* in cases of this group is organic heart disease directly or indirectly affecting a bundle branch. These cases are far more common than they are usually considered to be and in some the intermittent block is undoubtedly the precursor of permanent branch block. Consequently paroxysmal bundle-branch block which, like permanent bundle-branch block, may be the only evidence of organic heart disease, is of considerable significance, a fact which is not conveyed by the term "functional."

Association with Chronic Rheumatic Heart Disease.—Transient or intermittent bundle-branch block may occur less frequently in younger individuals with chronic heart disease of rheumatic origin. In all of these cases the periods of block have coincided with periods of poor myocardial function as manifested by cardiac failure, except in Case 5 in which such block is attributed to progressive fibrosis. The bundle and its branches are actually only specialized portions of the ventricular myocardium and it seems reasonable to assume that their function may be disturbed in the same way and at the same time as that of the myocardium.

Association With Acute Infections.—Transient bundle-branch block has been repeatedly attributed to acute infections and particularly to an active rheumatic process. Diphtheria on rare occasions can cause this type of transient conduction disturbance. Transient partial and complete A-V dissociation in otherwise clinically normal hearts is known to occur on rare occasions during acute infections, but we could find no conclusive evidence that acute infection, other than diphtheria, has directly caused a transient bundle-branch block.

With an active rheumatic infection in the presence of chronic rheumatic heart disease it is unjustifiable to attribute a transient bundle-branch block solely to a specific action of the acute process on the conducting tissue and to ignore the effects of the chronic lesion. In every case reported in which the transient branch block could be related in any way to an active rheumatic infection there was evidence of chronic valvular disease and poor myocardial function. After examining these cases critically it seems to us that there is more evidence to favor the opinion that infection usually acts largely through its effects on the whole myocardium including the bundle branches rather than specifically on a bundle branch or the blood supply of that branch. Rheumatic arteritis affecting a bundle branch may, however, be considered as a possible infrequent etiological factor as, perhaps, in Case 1 which is the best instance of a paroxysmal bundle-branch block being associated solely with an acute rheumatic infection. In Cases 2 and 10, although an infection was present, there was an obvious correlation between the branch block and dysfunction of the myocardium. In over 5,000 electrocardio-

grams taken at the House of the Good Samaritan in Boston on approximately 1,500 carefully followed patients with active rheumatic infection, no instance of transient bundle-branch block has been observed by Dr. T. Duckett Jones.

Association with Drugs.—Temporary bundle-branch block has been said to appear occasionally during the administration of drugs in the treatment of heart disease, particularly digitalis and quinidine. This has been shown to be the case with quinidine[50] but we could find no evidence that digitalis might have this effect.

Vagal Influences.—It is well known that the vagus nerves supply fibers to the S-A and A-V nodes and that carotid sinus pressure acting reflexly through the vagi may produce varying degrees of block in either node. The vagus has also been credited with the ability to depress conduction through the bundle branches.[13, 32, 39] The type of bundle-branch block described by Wolff, Parkinson, and White may be induced by carotid sinus pressure[39, 53, 57*] but, if the explanation of Holzmann and Scherf[23] and of Wolferth and Wood[56] for its mechanism be accepted, this is not a true bundle-branch block and increase in vagal tone must act on the A-V node inhibiting the transmission of impulses over the normal pathway and forcing their passage by way of the short-circuiting bundle. As regards true bundle-branch block we have been unable to find any published evidence that sustained inhibition of bundle-branch conduction can be produced by vagal stimulation, nor have we been able to induce such inhibition in our own cases in whom bundle-branch block was previously present (Cases 2, 3, 4, 8). During periods of marked S-A slowing or A-V dissociation produced by mechanical vagal stimulation or after the administration of acetyl-β-methylcholine occasional broad complexes may appear which have been interpreted as showing bundle-branch block.[13, 42] We believe that these are escaped beats of idioventricular origin because there is usually either absence of correlation between the P-waves and these wide QRS complexes or the P-R intervals are shortened, when in the presence of vagal stimulation unchanged or lengthened P-R intervals would be expected. However, in Case 8 on each of two occasions during marked S-A slowing without A-V dissociation produced by carotid sinus pressure single broad complexes occurred which were similar to those recorded during periods of bundle-branch block and whose P-R intervals were of the same length as those of the neighboring normal complexes (Fig. 12). For these reasons we believe that these two complexes may be examples of bundle-branch block rather than of ventricular escape. This provides evidence that the vagi may exert a direct influence upon bundle-branch conduction as is suggested

*The case reported by Wilson[53] displays the characteristics of the Wolff, Parkinson, and White type. Wilson found during the stimulation phase following the subcutaneous injection of atropine an alteration in the form of the QRS complex which he considered suggestive of branch block. There was, however, no increase in the Q-S interval.

by Daniélopolu and his associates[14] and Sigler[40] who found that intraventricular conduction may be slightly increased during carotid sinus stimulation.

It has been shown by animal experiments that when only a narrow strip of conducting tissue exists conduction is normal at slow rates but at rapid rates it is impaired.[5] Our results in Case 1, supported by the experience of others, suggest that intermittent bundle-branch block may be changed to normal conduction under vagal influences through the cardiac slowing induced by the increase in vagal tone. As in Cases 1 and 2 an increase in heart rate produced by a decrease in vagal tone or by any other means may in some cases be expected to produce branch block by further depressing the bundle branch.

A bundle branch may be partially damaged by progressive changes incident to myocardial failure or to inadequate metabolic exchanges resulting from poor coronary circulation to the conducting tissue, whether

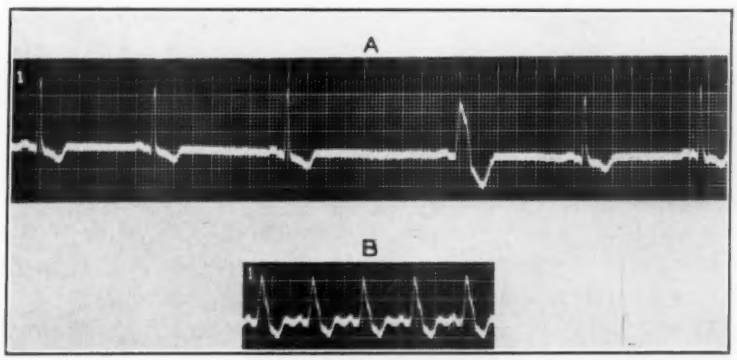

Fig. 12.—Case 8. *A*, (Jan. 15, 1937) single complex simulating left bundle-branch block occurring during right carotid sinus pressure which produced marked cardiac slowing. This record begins 9 sec. after the application and ends 6.5 sec. before the removal of the pressure. *B*, (Dec. 15, 1935) left bundle-branch block for comparison. Time-marker 0.20 and 0.04 sec.

there is partial fibrosis or not. Under such conditions the bundle branch may be able to conduct impulses within certain limits of rate but its conductivity will be impaired when the rate rises above a critical level. It is evident that the critical level will be dependent upon the degree of depression in the conducting fibers and it is not unreasonable to assume from the nature of the factors producing the inhibition that this may disappear or that the degree of depression may fluctuate or, in the case of fibrosis, progress. By accepting such a view it is possible to explain respectively transient bundle-branch block (Cases 4, 6, 8, 11, 12, 13), intermittent bundle-branch block (Cases 1, 2, 3, 5, 7, 9, 10) and intermittent progressing to persistent bundle-branch block (Cases 1, 5). In the routine records of our cases there was no *constant* correlation between the cardiac rate and the degree of intraventricular conduction. So long as the degree of depression may fluctuate it is not surprising that such a correlation was frequently not evident.

In the past emphasis has been placed upon the vagal influences when they were effective in converting bundle-branch block to normal conduction. In these cases, particularly, there has been the tendency to use the term "functional bundle-branch block." If heart disease is present or suspected these vagal influences, although dramatic, are not to be used as criteria in classifying the branch block. It should be clearly recognized that changes in the conducting tissue incident to organic heart disease constitute the fundamental and the significant cause of the paroxysmal bundle-branch block.

Mechanism.—The mechanism of this conduction disturbance has been discussed in previous reports and the terms "incomplete" or "partial" bundle-branch block applied.[21, 22, 45, 46, 47] A further consideration of the underlying mechanism seems desirable in the light of our present experience.

It is not necessary that complete block exist in one branch in order that impulses be forced to travel through the opposite branch and the myocardium to reach the affected ventricle. Conduction by this pathway may be necessary when one branch is only depressed. So long as the conduction time through the damaged branch is greater than that through the intact branch* plus the myocardial pathway between the two ventricles, the affected ventricle is activated by an impulse traveling through the latter channels, and bundle-branch block complexes result. Whenever the conduction time through the damaged branch passes the critical level and becomes shorter than that through the channel just mentioned, normal QRST complexes appear. It is therefore apparent that if conduction through the affected branch is close to this critical zone small changes in the conductivity of the depressed branch result in sudden and complete changes in the form of the ventricular complexes. Such small changes in conductivity result, for example, from the increase or decrease in diastolic rest due to slight alterations in heart rate as in Cases 1 and 2. In instances of 2 to 1, 3 and 4 to 1 bundle-branch block (Cases 26, 38, 46, 57), by a mechanism similar to that in partial A-V dissociation, the conduction of one or two impulses through the damaged branch increases the refractory period of that branch so that the succeeding impulse is delayed beyond the critical level and a branch block complex appears. The resulting rest permits the normal conduction of the next one, two, or three complexes.

When severe myocardial failure or coronary thrombosis is the cause of temporary bundle-branch block, conduction through the affected branch is severely depressed or even temporarily abolished. As improvement occurs the potential conduction time becomes progressively shorter

*It is known that lesions in both branches are frequently found in cases exhibiting bundle-branch block. The mechanism, however, would not be altered by damage or depression of both branches. Conduction in such instances would take place through the less damaged branch.

but branch block complexes remain until it passes below the critical level at which time normal complexes suddenly appear.

The complexes of the intermediate type which have been recorded during transitions, e.g., in Cases 2 and 10, probably occur when the conduction time through the damaged branch falls within the critical zone and is approximately equal to that through the intact branch plus the myocardium. The ventricle on the affected side is then partly activated by impulses which have passed along both routes.

SUMMARY

1. Due to the more common use of the electrocardiograph instances of paroxysmal bundle-branch block are becoming a more frequent finding. It is important to recognize the fundamental cause of the branch block in these cases and to differentiate clearly the inconsequential type from the group in which changes in the junctional tissue incident to heart disease are primarily responsible for the conduction disturbances.

2. The former group consists mainly of cases in which the QRS complex is wide and the P-R interval is abnormally short (Wolff, Parkinson, and White). This finding is no indication of heart disease.

3. Significant are the cases in which changes in the conduction tissue are a direct or indirect result of organic heart disease. Pathological processes, not necessarily permanent, in a branch of the bundle of His such as are associated with myocardial failure, coronary insufficiency, and coronary thrombosis, with or without partial fibrosis of a branch, are primarily responsible for transient or recurrent bundle-branch block. Although physiological changes may act as precipitating factors it should be recognized that these are effective only in the presence of actual changes in the conducting tissue. It is doubtful whether an acute infectious process other than diphtheria can cause transitory branch block; perhaps very rarely a rheumatic infection may so act.

4. Fifty-eight cases of paroxysmal bundle-branch block were found reported in the literature and to these we have added thirteen new cases. Of the seventy-one patients there was clear evidence of heart disease in sixty-five, of which thirty-five were males, twenty-nine were females, and one was of unrecorded sex. The six patients without other evidence of heart disease are excluded from the analysis although we consider the presence of paroxysmal bundle-branch block in itself evidence of heart disease. Coronary or hypertensive heart disease was present in forty-four patients while chronic rheumatic heart disease was present in six. The etiology of the heart disease in the remaining fifteen patients was diphtheria in four; thyrotoxicosis in three; congenital in one; and obscure in seven. Left bundle-branch block was present in sixty of the sixty-five patients. Moderate to severe heart failure was associated with the branch block in twenty-five patients.

5. The effects of changes in vagal tone were studied in six of our cases. Single bundle-branch block complexes were obtained during vagal stimulation on two occasions in one of these. Our experience and the published data show that increase in vagal tone does not produce sustained inhibition of bundle-branch conduction. Variations in vagal tone affect the conduction only by decreasing or increasing the cardiac rate to such an extent that the depressed branch becomes capable or incapable of transmitting impulses.

6. The mechanism of "partial" or "incomplete" bundle-branch block is discussed in the light of present experience.

7. We conclude that paroxysmal bundle-branch block (without a very short P-R interval) is as a rule a sign of serious heart disease, most often due to coronary sclerosis but in some cases associated with rheumatic heart disease, diphtheria, and factors that cannot clinically be ascertained. It is doubtless more common than it has been thought to be; serial electrocardiograms taken during myocardial (congestive heart) failure, coronary insufficiency, and elevated heart rates may reveal transient defects in bundle-branch conduction.

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A CORRELATION OF THE FLUOROSCOPIC, CLINICAL, AND POST-MORTEM FINDINGS IN 155 CASES OF ORGANIC HEART DISEASE*

BERNARD S. EPSTEIN, M.D., BROOKLYN, N. Y., AND
JOHN B. SCHWEDEL, M.D., NEW YORK, N. Y.

RADIOGRAPHY of the heart is a basic diagnostic procedure which complements and augments information derived from physical examination and electrocardiography. Many attempts to estimate the size of the heart accurately have been made by radiologists ever since teleroentgenography became an accepted procedure. Within a comparatively short time correlations were evolved between the cardiac silhouette and various heart lesions. The surface area of the heart and the various cardiac diameters were compared with standards obtained by similar determinations made on normal individuals. The correlations vary from the cardiothoracic ratio to the ratio between the surface area of the cardiac silhouette and the surface area of the body, or the body weight or height.[1]

The failing common to these procedures is that the heart is considered as a unit rather than as an organ composed of four interdependent chambers. Thus they are of value in determining enlargement of the heart taken as a whole but offer no reliable data concerning the status of the individual chambers. Inasmuch as the clinical importance of determining enlargement of the several heart chambers is assuming significant proportions in the study of cardiac dynamics, a method which may be useful should receive careful consideration.

Fluoroscopic examination of the heart in the right and left anterior oblique positions and the postero-anterior position is a reliable method based on the effects of enlargement of each chamber on the border-forming portions of the heart shadow as seen in the various projections. It also takes into consideration the relationship of the heart to the esophagus and mediastinal structures.

The method is essentially one which offers qualitative rather than quantitative data in the sense that the findings are expressed in degrees of enlargement rather than numerically.

Among those who laid the groundwork for fluoroscopic examination of the heart may be mentioned Assman, Dietlen, Bordet, Vaquez, Roesler, Parkinson, Nemet, and others.[2]

*From the radiologic service of Dr. A. J. Bendick, Montefiore Hospital.

Most observations have been based on small series of cases, not all of which were proven at autopsy. At the suggestion of the late Geza Nemet we undertook to correlate the autopsy and cardioscopic findings in a series of cases sufficient for numerical analysis.

MATERIAL

This series collected over a period of six years consists of 155 cases of organic heart disease each of which had been fluoroscoped shortly before death. Thanks are due the department of pathology for observations made so that a reliable comparison could be made of the fluoroscopic and the autopsy findings.

The cases are divided into two groups, one consisting of 88 cases of valvular heart disorders of rheumatic origin, the other of 67 cases of nonvalvular heart disease including the cases of hypertension and cardiovascular disease with arteriosclerosis. The group of congenital heart disease cases was omitted because it was too small to be of value here. No mention is made of the great vessels in this report.

Clinical observations were made by various members of the medical division. As a rule the clinical diagnoses were based on the criteria laid down by the American Heart Association.

The age and sex incidence is summarized in Table I.

TABLE I

AGE AND SEX INCIDENCE

AGE	VALVULAR HEART DISEASE		NONVALVULAR HEART DISEASE	
	MALE	FEMALE	MALE	FEMALE
0- 9	6	0	0	0
10-19	16	8	0	0
20-29	14	8	0	0
30-39	5	4	1	1
40-49	10	8	5	2
50-59	6	3	14	6
60-69	0	0	20	7
70-over	0	0	6	5

PROCEDURE

Fluoroscopies were done with the patient in the erect position. Observations were made in the right and left anterior obliques and the frontal positions. In each case the esophagus was visualized by means of barium paste* and the position of the trachea and main bronchi was determined. We did not use standard angles of obliquity since it was found that the optimum angle of visualization varied with the individual.

Chamber enlargements were recorded as maximal, moderate and no enlargement. No attempt was made to distinguish hypertrophy

*The barium paste was prepared by slowly adding water to precipitated barium sulfate while stirring vigorously. Enough water is added to make a fairly thick, smooth mixture. A small amount of chocolate syrup may be added for flavoring.

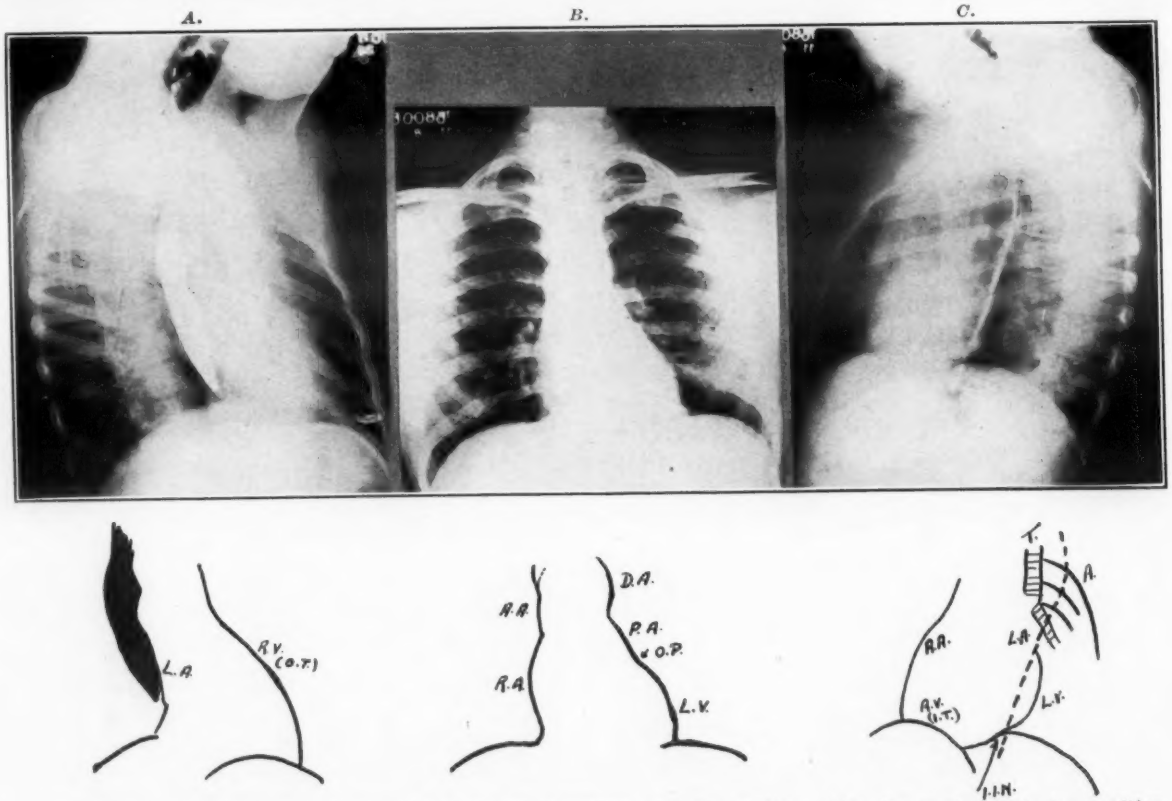

Fig. 1.—A normal heart, radiographed in the right anterior oblique position (*A*), the postero-anterior position (*B*), and the left anterior oblique position (*C*).

L.A., left auricle; *R.A.*, right auricle; *L.V.*, left ventricle; *R.V.* (*I.T.*), right ventricle, inflow tract; *R.V.* (*O.T.*), right ventricle, outflow tract; *O.P.* oscillating point (the point of opposite pulsation between the right ventricle and left ventricle at the junction of the second and third left cardiac arcs; *I.I.N.*, inferior interventricular notch between the left ventricle and inflow portion of the right ventricle; *A.A.*, ascending aorta; *D.A.*, descending aorta.

Note the straight outline of the barium-filled esophagus in the right anterior oblique position, indicating no enlargement of the left auricle. The relationship of the anterior aspect of the heart to the anterior chest wall as seen in Fig. 1*A* and the left ventricle to the spine as seen in Fig. 1*C* should also be noted.

Most observations have been based on small series of cases, not all of which were proven at autopsy. At the suggestion of the late Geza Nemet we undertook to correlate the autopsy and cardioscopic findings in a series of cases sufficient for numerical analysis.

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AGE	VALVULAR HEART DISEASE		NONVALVULAR HEART DISEASE	
	MALE	FEMALE	MALE	FEMALE
0- 9	6	0	0	0
10-19	16	8	0	0
20-29	14	8	0	0
30-39	5	4	1	1
40-49	10	8	5	2
50-59	6	3	14	6
60-69	0	0	20	7
70-over	0	0	6	5

PROCEDURE

Fluoroscopies were done with the patient in the erect position. Observations were made in the right and left anterior obliques and the frontal positions. In each case the esophagus was visualized by means of barium paste* and the position of the trachea and main bronchi was determined. We did not use standard angles of obliquity since it was found that the optimum angle of visualization varied with the individual.

Chamber enlargements were recorded as maximal, moderate and no enlargement. No attempt was made to distinguish hypertrophy

*The barium paste was prepared by slowly adding water to precipitated barium sulfate while stirring vigorously. Enough water is added to make a fairly thick, smooth mixture. A small amount of chocolate syrup may be added for flavoring.

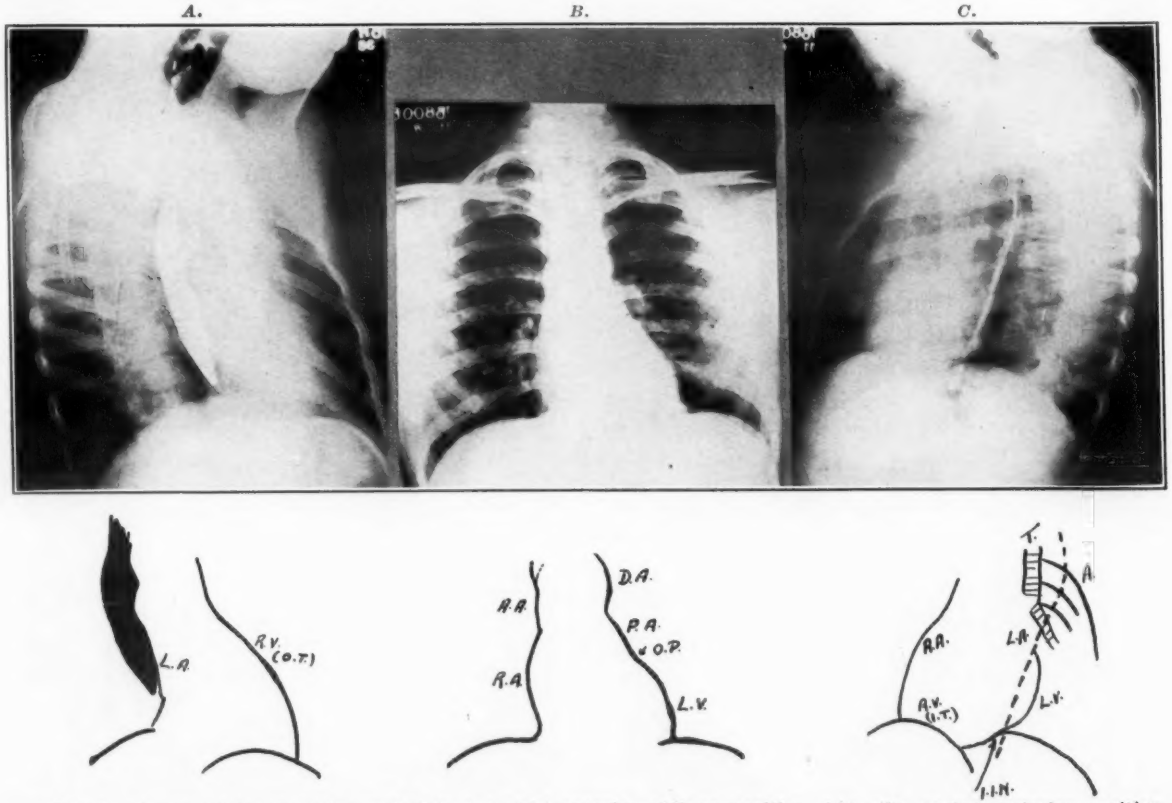

Fig. 1.—A normal heart, radiographed in the right anterior oblique position (*A*), the postero-anterior position (*B*), and the left anterior oblique position (*C*).

L.A., left auricle; *R.A.*, right auricle; *L.V.*, left ventricle; *R.V.* (*I.T.*), right ventricle, inflow tract; *R.V.* (*O.T.*), right ventricle, outflow tract; *O.P.* oscillating point (the point of opposite pulsation between the right ventricle and left ventricle at the junction of the second and third left cardiac arcs; *I.I.N.*, inferior interventricular notch between the left ventricle and inflow portion of the right ventricle; *A.A.*, ascending aorta; *D.A.*, descending aorta.

Note the straight outline of the barium-filled esophagus in the right anterior oblique position, indicating no enlargement of the left auricle. The relationship of the anterior aspect of the heart to the anterior chest wall as seen in Fig. 1*A* and the left ventricle to the spine as seen in Fig. 1*C* should also be noted.

from dilatation fluoroscopically. Post-mortem observations of the size of the cardiac chambers were made in similar degree, and the evidences of valvular defects, muscle hypertrophy, and coronary artery disease were noted.

The data also were analyzed from other viewpoints. In the rheumatic group the chamber enlargements associated with various valvular defects were tabulated. The accuracy of the clinical diagnoses was determined by comparison with the autopsy findings. Ventricular thickness, the incidence of congestive heart failure, auricular fibrillation, and pericarditis were also noted.

In the nonvalvular group the incidence of hypertension, coronary artery disease, and heart failure was correlated with chamber enlargement, murmurs, valvular defects, and muscle thickness. The comparative accuracy of the clinical diagnoses was determined.

FLUOROSCOPIC CRITERIA

The criteria for estimating chamber enlargement in this study are those submitted by Nemet and adopted for inclusion in the *Criteria for Cardiac Diagnosis.*[3] Additional refinements since evolved were taken into consideration.[4]

Briefly, the criteria are:

1. Left Auricle.—Protrusion of the left auricular shadow into the retrocardiac space displacing the barium-filled esophagus posteriorly, best seen in the right anterior oblique position. Deviation of the barium esophagus to the right in the frontal projection, found in more advanced cases of left auricular enlargement. Elevation and compression of the left main bronchus in the left anterior oblique position, indicative of vertical enlargement of the chamber. The presence of the right border of the left auricle just within the right cardiac border, or on the upper portion of the right cardiac border also may be determined fluoroscopically in the more advanced cases.

Moderate enlargement is believed present when posterior and horizontal enlargement is found. Maximal enlargement is indicated by the presence of posterior, horizontal, and vertical enlargement of more advanced degree.

2. Left Ventricle.—Increase in the length of the lower left cardiac arc as measured from the point of opposite pulsation downward. Projection of the apex below the diaphragm during deep inspiration. Posterior bulging of the left ventricle into the retrocardiac space and depression of the inferior interventricular notch in the left anterior oblique position.

Moderate enlargement is manifested by posterior bulging of the ventricle with downward apical displacement. Maximal enlargement

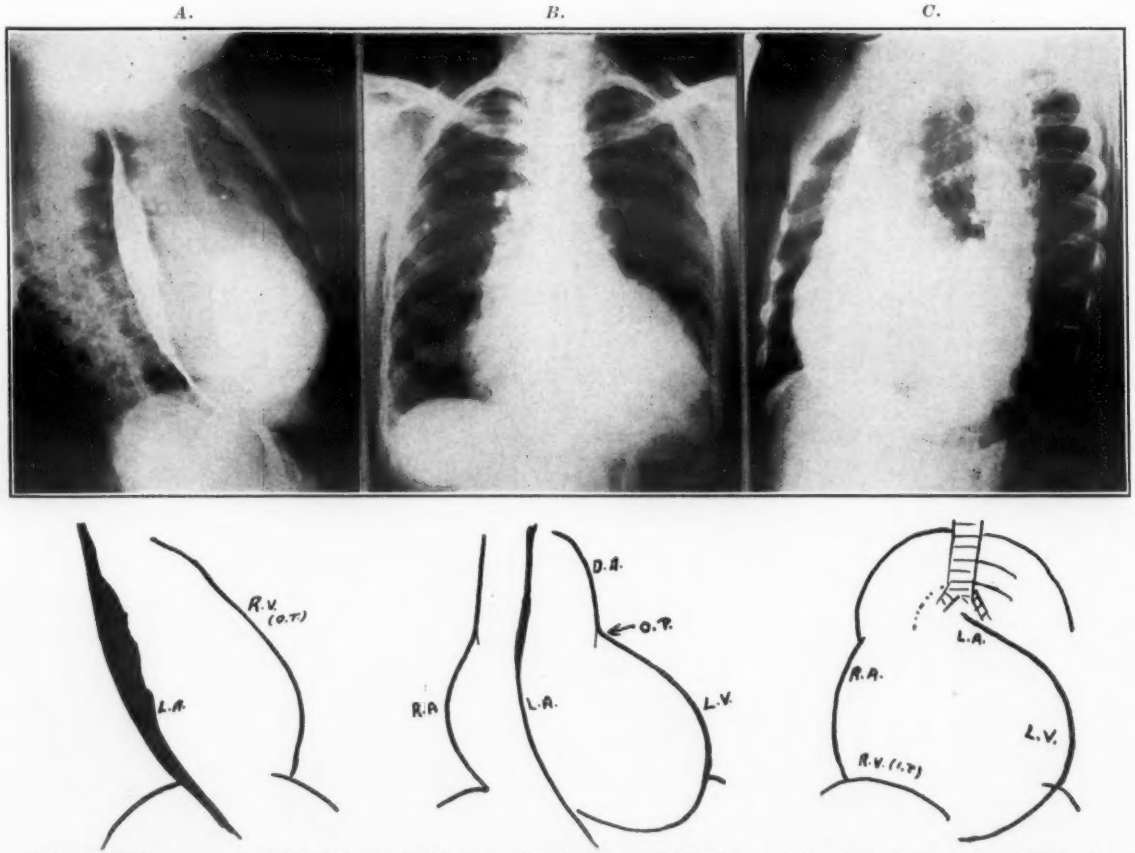

Fig. 2.—Enlargement of the left ventricle is maximal. The oscillating point is shifted upwards. Moderate enlargement of the right ventricle is demonstrable only in the right anterior oblique position, being obscured by the left ventricle in the anterior projection. The tremendous posterior bulge of the left ventricle is demonstrated in the left anterior oblique position.

implies massive posterior bulging and depression of the interventricular notch in the left anterior oblique view. The third left cardiac arc is lengthened, shifting the point of opposite pulsation upward.

3. Right Auricle.—Increase in the length of the right auricular appendix segment as seen in the left anterior oblique position. Increase in the acuity of the right atrioventricular angle in the left anterior oblique position. Increase in the posterior convexity of the right auricle in the right anterior oblique position so that the shadow of the barium esophagus passes through its opacity. This latter is indicative of enlargement of the body of the right auricle.[5]

Moderate enlargement is considered present when the right auricular segment is moderately increased and elevated. Maximal enlargement means enlargement of the auricular segment so that it assumes approximately a horizontal plane. When enlargement of the body of the chamber is present maximal enlargement is diagnosed.

4. Right Ventricle.—Increase in the length and convexity of the second left cardiac arc, depressing the point of opposite pulsation as seen in the frontal projection. Protrusion of the anterior aspect of the right ventricle into the retrosternal space in the right anterior oblique position (outflow tract).

Increase in the length of the diaphragmatic surface of the heart as measured from the right atrioventricular junction to the inferior interventricular notch as seen in the left anterior oblique position. Rotation of the right auricle upwards and posteriorly so that the right ventricle comes to form part of the lower right cardiac border in the frontal projection (inflow tract).

Moderate enlargement of the right ventricle is considered present when there is definite enlargement of the outflow tract. Maximal enlargement is diagnosed in the presence of both inflow and outflow tract enlargement.

CORRELATIONS OF THE RADIOSCOPIC, CLINICAL, AND POST-MORTEM FINDINGS

Valvular Group

1. Left Auricle (correlations possible in 88 cases).—Maximal enlargement was found at autopsy in 57 cases. Of these, 53 had been reported correctly fluoroscopically. The remaining four cases had been reported as moderately enlarged.

Moderate enlargement was found at autopsy in 20 cases. Eighteen of these had been reported correctly. Of the remaining two cases, one had been reported as maximally enlarged and one as not enlarged.

No enlargement was found in 11 cases, and was reported correctly in 10. The single case incorrectly diagnosed had been reported as moderately enlarged.

Fig. 3.—Enlargement of the left auricle deviates the barium-filled esophagus posteriorly and to the right. Enlargement of the outflow tract of the right ventricle is seen in both the right anterior oblique and postero-anterior projection. In the left anterior oblique position the left ventricle is seen to be of normal size, and the diaphragmatic extent of the right ventricle (inflow tract) is likewise not increased.

2. Left Ventricle (correlations possible in 88 cases).—Maximal enlargement was found at autopsy in 45 cases. Of these 44 had been reported correctly. The remaining case had been reported as moderately enlarged.

Moderate enlargement was found at autopsy in 33 cases and had been reported correctly in 27. Of the remaining six cases, four had been reported as maximally enlarged and two as not enlarged.

No enlargement was found in 10 cases and had been reported correctly in five. Of the remaining five cases four had been reported as moderately enlarged and one as maximally enlarged.

3. Right Auricle (correlations possible in 54 cases).—Maximal enlargement was found in 26 cases and had been reported correctly in 24. The remaining two cases had been reported as moderately enlarged.

Moderate enlargement was found in 18 cases and had been reported correctly in 14. The other four cases had been reported as maximally enlarged (two cases) and not enlarged (two cases).

No enlargement was found in 10 cases and was reported correctly in 10 cases.

4. Right Ventricle (correlations possible in 88 cases).—Maximal enlargement was found at autopsy in 53 cases and had been reported correctly in 48 of these. The remaining five cases had been reported as moderately enlarged.

Moderate enlargement was found in 27 cases and was reported correctly in 24. Of the remaining three cases two had been reported as maximally enlarged and one as not enlarged.

There were eight cases in which no right ventricular enlargement had been found. Seven of these had been reported correctly. The remaining case had been reported as moderately enlarged.

The percentage of accuracy of the qualitative estimation of left auricular enlargement was 92, of left ventricular enlargement 86, of right auricular enlargement 85, and of right ventricular enlargement 89. In most cases which had been wrongly diagnosed the mistake was one of degree—that is, very rarely was a case with no enlargement of a chamber considered to have maximal enlargement of that chamber, or vice versa. If this is taken into consideration, the incidence of correct diagnosis should be considered as greater than that mentioned above.

In checking the errors in diagnosis the following facts stand out; those cases incorrectly diagnosed as having maximal enlargement of the left ventricle and found to have moderate enlargement at autopsy were all cases in which the mitral stenotic element predominated. Maximal enlargement of the right ventricle was present in every case. Those cases incorrectly reported as having moderately enlarged left

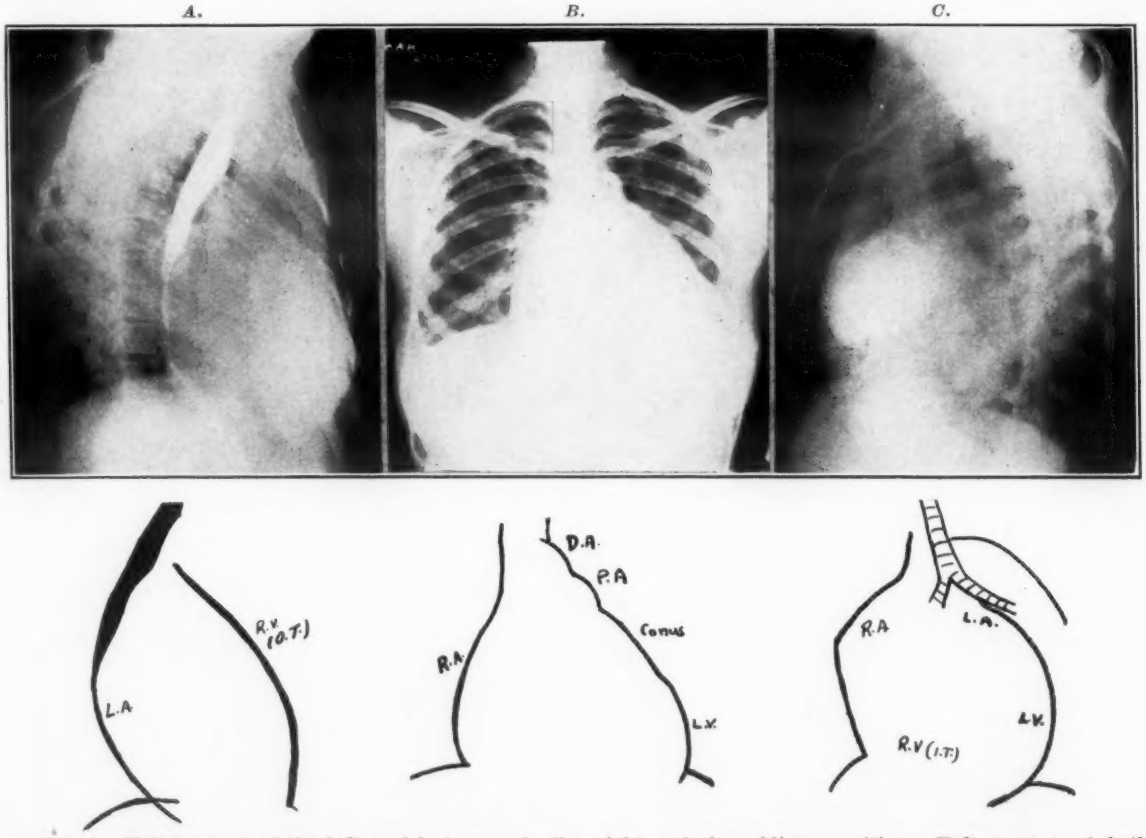

Fig. 4.—Enlargement of the left auricle is seen in the right anterior oblique position. Enlargement of both the inflow and outflow tracts of the right ventricle is present. The left ventricle is likewise moderately enlarged. Enlargement of the right auricular segment is seen in the left anterior oblique position.

ventricles and found to have no enlargement at autopsy likewise were found to have predominant mitral pathology with maximal right ventricular enlargement.

Enlargement of the right ventricle first results in an increased prominence of its conus portion anteriorly. This is followed by rotation towards the left and upwards as the natural restraints of the pericardium, sternum, spine, lungs, and remaining cardiac musculature exert their influence. As enlargement of the right ventricle progresses, the left ventricle is displaced posteriorly, so that in the left anterior oblique position the left ventricular segment seems to be larger than it actually is.

In the frontal plane maximal enlargement of the right ventricle may result in such enlargement of the transverse diameter of the heart that the left cardiac border, formed almost entirely by the right ventricle, approximates the axilla. In these cases the left ventricle may be represented by a small demilune low on the left cardiac silhouette. We have seen cases in which the entire left border of the heart was formed by the right ventricle, the left ventricle being rotated so that it assumed a completely posterior position.

For these reasons a diagnosis of left ventricular enlargement in the presence of maximal right ventricular enlargement should be made only after careful determination of the point of opposite pulsation in the frontal position to determine the length of the left ventricular segment and localization of the inferior interventricular notch in the left anterior oblique position. A diagnosis of left ventricular enlargement should not be made unless there is definite elongation of the third left cardiac arc, depression of the inferior interventricular notch, or bulging into the retrocardiac space.

Errors in the estimation of the size of the right ventricle were less frequent. In reviewing the cases diagnosed incorrectly it was noted that the cases of maximal enlargement wrongly diagnosed as moderately enlarged all occurred in hearts with marked aortic lesions (three cases with aortic insufficiency and two with aortic stenosis) with maximal left ventricular enlargement.

Massive enlargement of the left ventricle masks coincident right ventricular enlargement by shifting the point of opposite pulsation upward, thereby shortening the apparent extent of the second left cardiac arc. In the right anterior oblique position the left ventricular mass may overshadow the anterior bulge of the right ventricle and obscure outflow tract enlargement.

It would seem, then, that special attention should be given to the retrosternal bulge of the cardiac contour in the right anterior oblique position before estimating the size of the right ventricle in the presence of maximal left ventricular enlargement. The diaphragmatic extent of the right ventricle should likewise be noted carefully.

Examination of the patient in deep inspiration may help visualize the inferior interventricular notch. Quite often this landmark cannot be located definitely, and this should be taken into consideration before conclusions are reached.

Clinical-Pathological Correlations

A clinical diagnosis of congestive failure was made in 81 cases, all of which were confirmed at autopsy. Of the seven patients without heart failure, three had subacute bacterial endocarditis, two had chronic nephritis, one had advanced hypertension, and one was a six-year-old child with active carditis.

Subacute bacterial endocarditis occurred in 12 cases. The mitral and aortic valves were equally involved. Congestive failure occurred in nine instances.

Seven patients, all in the age groups above forty, had myocardial infarcts.

The incidence of valvular lesions together with the resultant chamber enlargements is summarized in Table II. These findings were derived from the post-mortem observations.

Mitral valvular pathology alone was found in 14 cases, and aortic valve pathology alone was found in 12 cases. A double mitral lesion with a single aortic lesion (most often insufficiency) occurred 21 times, while a double aortic lesion with a single mitral lesion occurred but once. Double aortic and double mitral lesions occurred in 15 individuals. Tricuspid stenosis and insufficiency were associated with the double aortic and double mitral lesions three times. Tricuspid insufficiency in association with other valvular defects occurred 25 times, while a double tricuspid lesion occurred six times. A single aortic and single mitral lesion occurred ten times. Endocarditis of the pulmonary valve which did not produce an anatomic defect was found in five cases, all of which had coexistent panvalvulitis.

The average thickness of the right ventricular myocardium in the cases considered maximally enlarged (55 cases) was 6.5 millimeters. In this group maximal enlargement of the left ventricle occurred 28 times, moderate enlargement 22 times, and no enlargement 5 times. The average thickness of the left ventricular wall in this group was 16.0 millimeters.

The average thickness of the right ventricular myocardium in the cases considered moderately enlarged (25 cases) was 5.6 millimeters. In this group maximal enlargement of the left ventricle occurred 15 times, moderate enlargement 10 times, and no enlargement once. The average thickness of the left ventricular wall was 17.2 millimeters.

The average thickness of the right ventricular myocardium in the cases with no enlargement (8 cases) was 3.2 millimeters. In this group

TABLE II

LESION	NUMBER OF CASES	DEGREE OF ENLARGEMENT													
		RT. VENT.				LT. VENT.				RT. AURIC.			LT. AURIC.		
		MAX.	MOD.	NO	VENT. (MM.)	MAX.	MOD.	NO	VENT. (MM.)	MAX.	MOD.	NO	MAX.	MOD.	NO
M. S., M. I., A. I.	10	8	2	0	5.9	8	2	0	16.9	5	2	0	9	1	0
M. S., M. I., A. I., T. I.	10	9	1	0	7.3	6	3	1	15.3	5	3	0	9	1	0
M. S., M. I., A. S., A. I.	8	2	5	1	5.7	4	4	0	18.6	1	3	2	3	2	3
A. I.	8	2	3	3	4.6	6	2	0	18.4	3	2	2	4	0	4
M. S., M. I., T. I.	6	5	1	0	6.3	3	2	1	15.5	3	1	0	5	1	0
M. S., M. I.	6	5	1	0	6.0	0	6	0	11.5	2	2	1	4	2	0
M. I., A. I.	5	2	2	1	5.4	5	0	0	18.6	1	0	2	2	3	0
M. I.	5	2	2	1	5.8	1	2	2	13.0	1	1	2	3	1	1
M. S., A. I.	4	2	1	1	7.7	1	3	0	15.2	2	1	0	1	3	0
M. S., M. I., A. S., A. I., T. I.	4	0	3	1	5.0	2	1	1	16.2	0	4	0	3	1	0
A. S.	3	2	1	0	7.3	3	0	0	17.3	1	1	0	2	1	0
M. S.	3	3	0	0	6.3	1	2	0	13.3	3	0	0	3	0	0
M. S., M. I., A. S., A. I., T. S., T. I.	3	3	0	0	6.0	2	1	0	17.0	3	0	0	2	1	0
M. S., T. I.	2	2	0	0	5.5	0	1	1	17.0	2	0	0	2	0	0
M. S., M. I., T. S., T. I.	2	2	0	0	5.0	0	1	1	16.5	1	1	0	2	0	0
M. I., T. I.	2	2	0	0	4.5	1	1	0	10.5	2	0	0	2	0	0
A. S., A. I.	1	0	1	0	4.0	1	0	0	20.0	0	0	1	0	1	0
A. S., A. I., M. I.	1	0	1	0	5.0	0	1	0	15.0	0	0	1	0	0	1
M. S., A. I., T. I.	1	1	0	0	6.0	0	1	0	13.0	1	0	0	1	0	0
M. S., M. I., T. S., T. I., A. I.	1	1	0	0	7.0	0	1	0	15.0	0	0	0	1	0	0

maximal enlargement of the left ventricle occurred three times, moderate enlargement twice, and no enlargement three times. The average thickness of the left ventricular wall was 16.2 millimeters.

The heaviest heart weighed 1,400 grams, the lightest 320 grams. The average weight was 750 grams. There did not seem to be any definite relationship between the heart weights and the patients' ages in this series. No definite relationship could be found between the valvular lesions and heart weights, although the various valvular defects seemed to produce constant changes in the sizes of the chambers. For instance, one heart weighing 1,050 grams had a double mitral lesion with tricuspid insufficiency, while a heart weighing 1,200 grams had an isolated aortic insufficiency. Still another heart with an isolated aortic insufficiency in a patient of the same age weighed but 400 grams. The heart weighing 1,400 grams had mitral stenosis with aortic insufficiency (Table III).

TABLE III

VALVE DEFECTS	HEART WEIGHTS (GM.) AND AGE OF PATIENT (YR.)			
M. S., M. I., A. I.	630 (16)	500 (38)	550 (29)	700 (17)
	520 (44)	500 (16)	600 (22)	
	450 (13)	560 (24)	350 (46)	
M. S., M. I., A. S., A. I.	550 (21)	550 (21)	480 (24)	
	480 (60)	480 (56)	400 (28)	
	450 (47)	730 (24)		
M. S., M. I., A. I., T. I.	550 (9)	400 (14)	550 (19)	
	1200 (17)	550 (29)		
	400 (12)	400 (24)		
A. I.	1000 (36)	540 (34)	730 (52)	
	680 (52)	850 (38)	850 (46)	
	360 (10)	400 (14)		
M. S., M. I., T. I.	450 (39)	600 (22)		
	360 (13)	1050 (20)		
	640 (44)	500 (18)		
M. S., M. I.	800 (52)	600 (7)		
	500 (21)	300 (48)		
M. I., A. I.	950 (22)	500 (11)	660 (15)	
	480 (47)	1100 (18)		
M. I.	500 (11)	550 (45)	350 (36)	
	320 (23)	290 (5)		
M. S., T. I.	310 (12)	720 (47)		
M. S., A. I.	290 (14)	1400 (22)		
M. S., M. I., A. S., A. I., T. I.	450 (49)	410 (34)		
	700 (17)	480 (40)		
A. S.	530 (52)	890 (46)	530 (52)	
M. S.	300 (28)	550 (40)	660 (42)	
M. S., M. I., A. S., A. I., T. S., T. I.	600 (21)	660 (23)		
M. S., M. I., T. S., T. I.	550 (24)			
A. S., A. I.	320 (46)			
A. S., A. I., M. I.	450 (54)			
M. I., T. I.	750 (13)	580 (8)		
M. S., A. I., T. I.	600 (22)			
M. S., M. I., T.S., T. I., A. I.	350 (14)			

Pericarditis was found in 38 cases at autopsy and was diagnosed during life in 12 cases. The poor clinical showing may be due to the fact that the 38 cases included both old and recent pericarditis, and

TABLE IV

FINDINGS	NUMBER OF CASES	DEGREE OF ENLARGEMENT													
		LT. VENT.				RT. VENT.				LT. AURIC.			RT. AURIC.		
		MAX.	MOD.	NO	VENT. (MM.)	MAX.	MOD.	NO	VENT. (MM.)	MAX.	MOD.	NO	MAX.	MOD.	NO
Hypertension, coronary artery disease, heart failure	28	22	6	0	17.3	6	16	6	5.5	3	15	8	3	11	7
Coronary artery disease, heart failure	13	2	9	2	14.0	2	6	5	5.6	0	3	9	0	1	6
Hypertension, coronary artery disease	8	5	3	0	17.4	0	5	3	4.3	0	3	5	0	1	4
Hypertension	8	4	4	0	20.5	1	1	6	4.2	0	2	6	0	1	7
Hypertension, coronary artery disease	4	3	1	0	21.0	1	3	0	6.2	0	3	1	0	3	0
Normal	4	0	1	3	12.0	0	0	4	3.0	0	0	4	0	0	4
Coronary artery disease	2	1	1	0	13.5	0	0	2	3.5	0	0	1	0	0	1

the patients may have been seen only during a quiescent stage. Twenty cases occurred in patients over twenty years old and 18 in patients under twenty. There was no definite predominance of any valvular lesions coincident with pericarditis.

There was complete agreement between the clinical and autopsy diagnoses in 32 cases. In 30 cases there was partial agreement (by partial agreement we mean that the valves involved were correctly diagnosed, but an error was made in the estimation of the presence or absence of either insufficiency or stenosis. For instance, a partial error was made in a case where a double mitral lesion with aortic insufficiency was diagnosed clinically and mitral and aortic insufficiency only was found at autopsy). In 16 cases the clinical diagnosis included more pathology than actually was present, and in 14 cases a valvular defect was omitted in the clinical diagnosis.

In 26 cases a complete error was made (by this we mean that a valvular defect was completely misdiagnosed. For instance, in a case where a double mitral lesion and aortic insufficiency was diagnosed during life, only an aortic insufficiency was found at autopsy).

Nonvalvular Group

This series consists of 67 cases. The incidence of hypertension, coronary artery disease and congestive failure, together with chamber enlargements found at autopsy is summarized in Table IV.

CARDIOSCOPIC POST-MORTEM CORRELATIONS

1. *Left Auricle* (correlations possible in 54 cases).—Maximal enlargement occurred in four cases and was estimated correctly fluoroscopically in three. The remaining case had been reported as moderately enlarged.

Moderate enlargement was found at autopsy in 21 cases and had been estimated correctly in 19. The remaining two cases had been reported as not enlarged.

No enlargement was found at autopsy in 29 cases and had been reported correctly in each instance.

2. *Left Ventricle* (correlations possible in 65 cases).—Maximum enlargement was found at autopsy in 37 cases and had been estimated correctly in 34 cases. The remaining three cases had all been reported as moderately enlarged.

Moderate enlargement was found at autopsy in 24 cases and had been reported correctly in 22. The remaining two cases had been estimated as maximally enlarged.

No enlargement was found at autopsy in five cases and had been correctly estimated in four. The remaining case had been reported as being moderately enlarged.

3. *Right Auricle* (correlations possible in 39 cases).—Maximal enlargement was present in three cases at autopsy and had been reported correctly in one. The other two cases had been reported as being moderately enlarged.

Moderate enlargement was found in five cases and had been reported correctly in three. The other two cases had been reported as not enlarged.

No enlargement was found in 31 cases and had been reported correctly in 30. The remaining case had been reported as moderately enlarged.

4. *Right Ventricle* (correlations possible in 63 cases).—Maximal enlargement was found in nine cases, seven of which had been correctly reported. The remaining two cases had been reported as moderately enlarged.

Moderate enlargement was found in 32 cases, 22 of which had been reported correctly. The remaining 10 cases had been reported as not enlarged.

No enlargement was found in 22 cases and was correctly reported each time.

The percentage of accuracy of the fluoroscopic estimation of the size of the left auricle was 96, of the left ventricle 92, of the right auricle 87 and of the right ventricle 80. Most of the errors were errors of degree, a finding similar to that in the valvular series.

From these figures one may assume that the accuracy of the determination of the size of the left auricle and left ventricle in nonvalvular heart disease is reliable. The figures on the right auricle are meager, and while conclusions should not be drawn, they seem to indicate that right auricular enlargement may be gauged with a fair degree of accuracy.

The accuracy of the estimation of the size of the right ventricle in this group is not as satisfactory as that for other chambers or for the right ventricle in the valvular group. In reviewing the cases in which mistakes occurred, one is impressed with the fact that all ten errors occurred in cases where moderate enlargement of the right ventricle was reported as no enlargement, and that almost all occurred in patients with left ventricular enlargement (eight with maximal enlargement, two with moderate enlargement).

The reason for the mistaken diagnoses is the same as mentioned in the discussion of similar errors in the valvular group, namely that enlargement of the left ventricle increases the extent to which the left ventricle occupies the left lateral border and diaphragmatic portion of the heart shadow, shortening the second left cardiac arc and protruding into the retrosternal space. The reason the error in right

ventricular estimation was greater in this group is that the incidence of maximal left ventricular enlargement is more frequent than in the valvular group.

CLINICAL-PATHOLOGICAL CORRELATIONS

In reviewing the figures in Table IV it is noted that the cases with hypertension alone and hypertension with coronary artery disease (22 cases) have a lower incidence of right ventricular enlargement than those cases in which cardiac insufficiency had supervened (45 cases). The average thickness of the right ventricular myocardium is greater in the cases with heart failure also. It would seem, then, that the presence of heart failure is one of the important factors in the production of the right ventricular hypertrophy in this series of cases. A corollary to this statement may be that the presence of definite right ventricular enlargement is presumptive evidence for cardiac insufficiency, not necessarily present when the chamber enlargement is first noted.

Left auricular enlargement also was more frequent in cases with heart failure.

The cases with hypertension alone or hypertension with heart failure have heavier left ventricular myocardiums than the cases with hypertension and coronary artery disease. This finding raises the question as to the relationship between cardiac hypertrophy and existent vascular supply.

Aneurysmal dilatation of the left ventricle was found at autopsy in nine cases. A correct diagnosis was made fluoroscopically four times. In four of the cases in which the diagnosis was not made the aneurysm involved the posterior aspect of the left ventricle, and in one case the right ventricle.

Definite murmurs were audible in 41 cases. Of these, a rough systolic apical murmur was heard in 7 cases, a soft apical systolic murmur in 22 cases, a generalized precordial systolic murmur in five cases, and a basal systolic murmur in two cases. A basal systolic and diastolic murmur was heard in five cases.

Definite valvular defects were found at autopsy in 10 cases. Four of these were aortic insufficiency on a luetic basis, one a mitral stenosis, two mitral insufficiency, one mitral stenosis and insufficiency, and one mitral insufficiency with tricuspid insufficiency.

The clinical diagnoses and post-mortem findings were in good agreement so far as the presence of coronary artery disease, cardiac infarction, and heart failure was concerned.

CONCLUSIONS

1. Cardioscopy is a satisfactory procedure for the qualitative estimation of the degree of enlargement of the individual heart chambers.

2. A series of 155 cases of valvular and nonvalvular heart disease is analyzed numerically from the viewpoint of chamber enlargements, valvular defects, ventricular hypertrophy, murmurs, cardiac failure, and the accuracy of clinical diagnoses as compared with autopsy findings.

3. Errors in estimating the size of either ventricle are more frequent when maximal enlargement of the other chamber is present. Certain precautions in avoiding these errors are mentioned.

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THE POSSIBILITY OF EMBOLI FROM ARTERIAL THROMBOSIS SHORT-CIRCUITING PERIPHERAL AND PULMONARY CAPILLARY CIRCULATIONS

Report of a Suggestive Case

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FOR years the medical fraternity in dealing with arterial thrombosis has had a sense of security from embolism in such lesions owing to the protection of the capillary network through which presumably emboli could not pass.

Certain unusual features are presented in the case abstracted below which suggest that partial revision of this view may be necessary.

A man sixty-eight years of age who had previously been in excellent health was suddenly seized with an attack of pain on exercise in both feet and legs. His previous vascular condition, as far as could be determined by physical examination, cardiogram, and x-ray, had been excellent and he had played hard tennis within four weeks of the onset of his illness, in itself a fair test of both cardiac reserve and circulation in his legs, though accidents have been known to occur in such conditions without previous symptoms.

His symptoms increased gradually for a month, with progressive disability until the date of his hospital admission on which he showed evidence of thrombosis of the arteries in both lower extremities probably on an arteriosclerotic basis. The lesion was more extensive on the left where the tibial artery was involved than on the right where only the arteries of the foot were affected. The extent of the lesion was demonstrated by the oscillometer and thermometric observations. Within a few days both femoral arteries were also involved. His vascular condition, other than his legs, showed no demonstrable change. Cardiograms had been taken in 1924, 1933, and 1934 and except for lessened amplitude in T_3, which in August of 1935 was almost isoelectric, were unchanged. These changes are certainly not unusual in a man of his age. The x-ray film of the heart could be superimposed on those taken on many occasions in the past ten years and there was no calcification of the aorta, while the femoral and tibial arteries showed only slight calcification.

His physical examination was negative except for the vascular condition of the legs. The retinal arteries showed no change.

His blood count was as follows: Hemoglobin, 100 per cent; Red blood cells, 4,830,000; White blood cells, 7,500; Polymorphonuclears, 59 per cent; Lymphocytes 38 per cent; Monocytes, 1 per cent; Basophiles, 1 per cent; Eosinphiles 1 per cent.

Urine was negative.

Thorough examination showed no demonstrable focus of infection, though for some years he had had a slight Vincent's infection of the gums, which was under control at the moment.

Three weeks previous to the onset of his leg symptoms he had had a febrile attack with gastric and abdominal symptoms, lasting a week, of unknown causation.

History, except for the excessive use of tobacco, was excellent.

To save his leg, treatment by the Pavex Boot, with its intermittent negative and positive pressure phases, was instituted, with marked improvement in the local circu-

lation. On September 7 he began to have a little abdominal pain, with right sided costovertebral tenderness, and a little temperature, leucocytosis of 8,000 with 70 per cent polys, which rose on the eighth to 12,000, with 83 per cent of polynuclear cells. There was no pus in the urine.

On September 10 he had a little tingling in the left arm and a little ataxia in the finger to nose test. He was seen by a neurologist who found no sign of central involvement. This, however, went on to a definite left-sided ptosis with very slight speech defect and was followed by tremor and weakness of the left arm, which was obviously central. On the eleventh of September he had a severe attack of upper abdominal distress with nausea and vomiting. In the absence of organic intrinsic lesions of the stomach, gallbladder, or upper intestine, which were ruled out by x-ray examination, we began to think in terms of either extension of his thrombosis to the abdominal vessels or a coronary thrombosis without precordial pain. The latter proved to be the case as shown by the cardiogram of September 12.

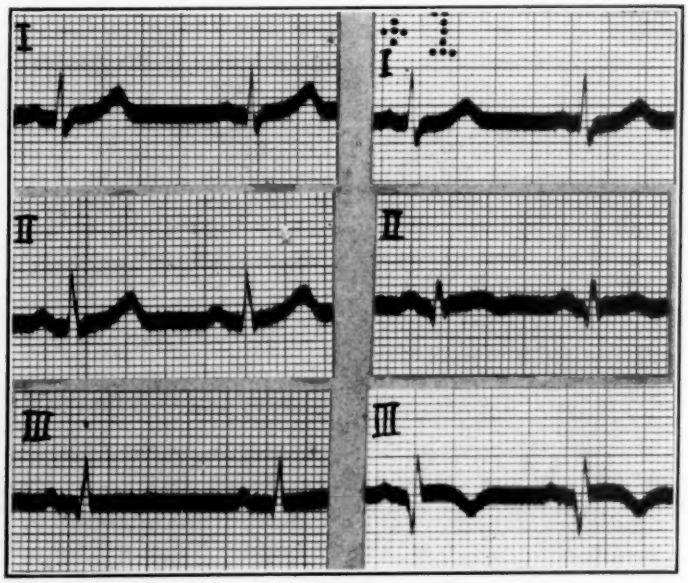

Fig. 1.

Fig. 2.

On September 24 he developed a new focus of clotting, the radial artery in the right arm. The symptoms of this lasted only a few hours.

For years he had had a low rather than high blood pressure, 112/70 and 115/80 in 1934. Blood pressure on admission was 120/62 but rose with the increase of his thrombosis to averages of 160 to 180 systolic and 70 to 90 diastolic, where it had remained until June, 1937.

Four months later, treatment still being continued, he had a second coronary attack. The causation of his initial lesion and of his numerous arterial accidents was puzzling to us. There was no evidence of anaphylaxis or infection. With each coronary occlusion he had a rise in temperature, leucocytes, and sedimentation rate. His uric acid had tended to be high for a number of years; 5.6 mg. per 100 c.c. in 1934; 4.8 mg. per 100 c.c. in 1933. On Sept. 12, 1935, blood urea was 0.36 gm. per liter; blood Nonprotein nitrogen, 0.33 gm. per liter; blood carbon dioxide combining power, 50 per cent. On September 26 blood cholesterol was 208 mg. per 100 c.c. On October 8 showed blood amylase 23.8; October 22, blood urea was 0.38 gm. per liter; blood cholesterol 225 mg. per 100 c.c. and blood uric acid 3.0 mg. per 100 c.c.

However, the tobacco and an unknown infection seemed to be the most reasonable precipitating causes in a man with only very slight demonstrable arterial changes. We could find no evidence of infection elsewhere and were inclined to think of the explanation as slow and generalized endarteritis, though we called the cause X-factor.

The treatment by the boot method was discontinued in the spring of 1936. Since that time he has had no further arterial accidents.

Having noticed in the literature reference to the occurrence of arteriovenous anastomoses, it seemed possible to the author that the alternating negative and positive pressures of the boot might have dislodged thrombi existing in the arterioles or in the Suquet-Hoyer canals. A discussion of the literature on these vessels may be pertinent in a theoretical discussion of the case.

The arteriovenous anastomosis first described by Hoyer[1] in 1877 was considered a very exceptional formation and of importance only in giving rise to the glomus tumor. The universality of its occurrence in the digits of the feet and hands and its functional importance in protecting extremities from cold has been proven by Grant[2] and by German observers[3] and the presence of thrombi in the anastomoses themselves as occurring regularly in thrombo-angiitis obliterans has been demonstrated by Popoff.[4] We may well pause and consider the possibility of an embolus becoming detached, passing into the veins, the heart, and the lungs. The problem is the probable clinical effect. In this a consideration of the size of the anastomoses is important. Grant gives the diameter as from 18 to 150 microns. Popoff,[4] however, states that in thrombo-angiitis obliterans abnormal functionless anastomoses are found which are much larger, 100 to 350 microns in diameter. The number of normal anastomoses is given by Grant as 50 to 500 per square centimeter. Except possibly from thrombosis in these abnormal anastomoses a lesion in the lung caused by embolism nonseptic in nature would be difficult if not impossible to detect clinically. The rich pulmonary blood supply would probably prevent increase in size of the small initial lesion. The result, then, of an embolus passing from the periphery into the lungs through arteriovenous anastomoses is probably, from a clinical point of view, relatively unimportant in noninfectious thrombosis. In a septic thrombosis, however, this might well give a clue to the origin of certain unexplained septic lesions in the lung.

Does this, however, complete the picture? The pulmonary capillary system still protects the arterioles of the general system from embolic processes. Are there any means by which the capillaries of the lesser circulation may be short-circuited? Olkon and Joannides[5] by direct observation demonstrated capillary vessels of two distinct calibers in the network of the capillary bed. Daly comments, "In this observation we have evidence that the blood in a number of the smaller capillaries covering the alveolar surface can be shunted through other channels." MacGregor[6] also suggests that a vascular shunt exists, in his work with drug

injection into isolated lungs. Wearn's repetition[7] of Thebesius' experiment[8] has shown a direct connection other than through the capillaries between the coronary sinus and the heart chambers. "Thebesius introduced his blowpipe into the coronary vein and observed the bubbles escaping into the immersed chambers. In this study the experiments of both Vieussens and Thebesius have been confirmed. Moreover, when perfusion was carried out through the coronary sinus at pressures ranging from 50 to 150 mm. Hg almost all of the perfusate ran out through the Thebesian vessels, and in only a few instances did a few drops escape through the coronary arteries. The result was obtained when saline, acacia, agar, and gelatin were used. Again in these hearts the capillaries were not injected thus showing that the perfusate must have escaped by a more direct and larger communication between the veins and the Thebesian vessels, without having passed through the capillaries." Finally Daly[9] in his recent Harvey lecture arguing from a teleological viewpoint based on arterial pressure in the lesser and the bronchial circulations, from nerve distribution and nerve stimulation, and from the fact that it is known that blood is not always circulating through the whole of the pulmonary vascular bed, reaches the following alternative conceptions:

First: A conception of a pulmonary vascular bed "in which a number of arteriole capillary vessel networks exist in parallel, so that arteriole constriction of one network diminishes the blood flow in the network it supplies, but shunts the blood through the remainder. This would not be effective in opening up all the alveolar capillaries, which is the desired result during muscular activity."

Second: He next applies the hypothesis of direct arteriovenous anastomoses mentioning that Grant proved in the rabbit's ear their direct control by the sympathetic nervous system and suggests that this hypothesis best conforms to the needs of the body for oxygenation during exercise. "The existence of such a mechanism would largely overcome the difficulties of correlating pulmonary sympathetic vasoconstrictor action with muscular exercise. Under resting conditions a certain number of the communication channels would be open and the alveolar capillary bed which they shunt, closed, whereas during muscular exercise the concomitant stimulation of the pulmonary sympathetic nerves would close the communicating channels and divert the blood through the capillaries, so increasing the vascular bed available for the uptake of oxygen."

Fat embolism, clinically, has been known to pass the peripheral capillaries and pulmonary capillaries and manifest itself in peripheral lesions. (See "Embolism," Nelson Loose-Leaf Living Medicine, Vol. 4, page 590.)

The existence of anastomoses between artery and vein in the lesser circulation would remove the protection of the capillaries of the lesser circulation and an embolus, originating in the arterioles of the foot for

example, might short-circuit both peripheral and pulmonary circulations and cause a lesion in the terminal arterioles of the peripheral circulation.

Would this be clinically manifested? In certain situations this probably can be answered in the affirmative, as in terminal arterioles of the coronary and cerebral arteries for example. The obvious criticism is that such a lesion is too small to be clinically recognized. While true of lesions in most locations it is suggested that even in a microscopic lesion the block of the arteriole and the consequent surrounding edema might be recognizable by electrocardiogram if located in certain of the coronary vessels. Any arteriole block would be followed by a thrombosis in the stagnant blood stream proximal to it, at least as far as the nearest proximal branch and possibly much further, with an ever widening radius of encircling edema. Such a lesion might well cause clinical subjective symptoms as well as certain electrocardiographic changes should the initial lesion be in the coronary arterioles.

The short-circuiting then of the peripheral capillary circulation for small emboli through arteriovenous anastomoses is certainly a physical possibility. The short-circuiting of the pulmonary capillary circulation by the means of these anastomoses is as yet hypothetical. A direct short-circuiting of the entire pulmonary circulation has been proven by the experiments of Thebesius and Wearn.[8] To offer an open pathway to the actual passing of an embolus through this pathway, however, is dependent upon pressure considerations of which too little is known.

As an alternative suggestion to the generalized endarteritis as a cause for the patient's numerous arterial accidents, I am presenting the possibility of dislodgment of thrombi proven by Popoff[4] to exist in at least one of the obliterative diseases, thrombo-angiitis obliterans. Such emboli, the dislodgment of which would certainly be aided by the intermittent negative and positive pressure, might, as sketched, short-circuit the peripheral capillaries through the arteriovenous anastomoses and the pulmonary circulation short-circuit the capillaries through the pulmonary arteriovenous anastomoses or the entire pulmonary circulation through the Thebesian vessels.

So far we have shown a possible path based only on physical or mechanical considerations by which emboli dislodged from a peripheral arteriole might short circuit both peripheral and pulmonary circuits and again lodge in the periphery. If, however, the element of infection be considered, the possibilities of passage are increased. Clumps of bacteria or even small particles of a diameter less than two red cells would readily pass the capillaries in both greater and lesser circulations and hence cause the peripheral lesions observed in the above quoted case.

It cannot be denied, however, that a conception of a generalized endarteritis may be the solution of the etiological problem and that the unknown infection from which the patient suffered a month prior to the onset of his symptoms may have resulted in the lesion in his leg and

also in his coronary and cerebral arteries. It is, however, significant that these accidents to the coronary and cerebral arterioles occurred only during the period of treatment with the intermittent pressure boot and that since the discontinuation of this treatment fifteen months ago, he has had no further accidents. It is, therefore, suggested that the dislodgment, by the treatment itself, of emboli which passed through anastomoses of both peripheral and pulmonary circulations, may have caused his numerous arterial accidents. If the infectious idea be accepted, the intermittent pressure might as readily have sent infected particles through the capillary circulations.

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THE DETERMINATION OF EXERCISE TOLERANCE BY THE TWO-STEP TEST*

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THE diagnosis of heart disease involves a consideration of etiology, anatomy, physiology, and the ability of the patient to perform physical exertion. Fairly satisfactory criteria have been established for the interpretation of the anatomical and physiological abnormalities that a patient may present, but many complications are encountered when an estimate of the exercise tolerance or functional capacity is attempted. The reserve power of the myocardium will determine in a large degree the amount of exertion that an individual can accomplish, although there are other factors, such as age, sex, muscular development, coordination, and reflex vascular responses, that affect the exercise tolerance. Furthermore, the ability to perform physical exertion may vary from day to day in the same individual and is influenced by such factors as the general well being, sleep, pain, psychic stress, diet, atmospheric conditions, and probably many others.

To define a standard of accomplishment for any particular group of individuals is obviously difficult, if not impossible, and such a standard must necessarily have a wide range of variation for normal individuals. Practically, it is not difficult to classify patients as to their functional capacity for clinical purposes, if they present obvious signs or convincing symptoms of heart failure while at rest, or on slight exertion. Not infrequently, however, patients in whom no abnormalities are discovered either by physical examination or by other diagnostic methods complain of symptoms suggestive of heart disease. This group is a particularly important one to those practicing medicine in cases where compensation is involved. In the age distribution of patients seen in Veterans' Administration Facilities, arteriosclerotic heart disease without definite physical signs must be considered and distinguished from the so-called functional heart disease and other noncardiac illnesses. Any method that will aid in demonstrating the presence or absence of myocardial or coronary insufficiency is therefore highly desirable.

Many tests have been proposed for estimating the exercise tolerance or functional capacity depending upon some strain, such as the physical

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effort of riding a bicycle to produce abnormal physiological responses characteristic of heart failure. Since these phenomena vary considerably, the criteria have been unsatisfactory and many of the tests are technically difficult and, therefore, limited in their practical usefulness. It has long been known that the blood pressure and pulse rate increase during work, but it has not been determined satisfactorily whether the blood pressure responses, which occur during exercise, are due to the increased output of the heart or to increased peripheral resistance. Wiggers[1] has demonstrated the type of blood pressure and pulse rate changes usually seen during exercise, by means of a circulatory schema, in which the heart rate and systolic discharge are increased and the peripheral resistance then lowered. In the human subject, however, the regulation of blood pressure is complicated by psychic reactions, reflexes from the sino-aortic region, hormonal constrictor and dilator substances, and probably a different response in various parts of the vascular system at the same time.

Many attempts have been made to obtain a measure of the functional capacity of the heart by observing the blood pressure and pulse rate before and after various types of exercise. Of the many modifications that have been proposed, the one described by Master and Oppenheimer[2] and by Master[3] appears to have certain advantages for routine determination of the exercise in ambulatory patients. The apparatus required is simple and easily available; the exercise is one that most individuals are accustomed to, without training; the amount of work performed can be roughly measured and tables of standard exercises, which take into consideration the age, sex, and weight of the individual, are available.

In the Master test the resting blood pressure and pulse rate are compared with the values for these phenomena two minutes after completing the exercise, which consists of repeated ascents on two steps, each nine inches high, a standard number of times in one and one-half minutes. If the levels for the blood pressure and pulse rate do not differ from the resting level, by more than ten points, the patient is considered to have a normal exercise tolerance. It is proposed to report our experience with the use of this test clinically and in addition some observations on the specificity of the criteria.

METHOD

One hundred patients were examined who presented symptoms suggestive of heart disease, the presence of which was subsequently confirmed in some cases by objective evidence, while in a larger group no definite signs of heart disease could be found. Some of the latter group were suffering from conditions that might influence the general tolerance to exercise.

In all cases a complete medical history was taken and a physical examination performed, after which the patient was allowed to lie on the examining table without being disturbed for at least thirty minutes, until the blood pressure and pulse rate seemed to be constant. Blood pressures were taken on the left arm using the auscultatory method and a mercury manometer. Diastolic pressure was read at the point where the sounds became very faint just before completely disappearing. On subsequent tests the preliminary history and physical examination were not repeated and no patient was tested who presented obvious signs of failure. Several patients were examined whose chief complaint suggested the anginal syndrome, but the exercise, required of them for a routine examination, did not precipitate any typical attack of pain.

The pressor reflex, according to the technique of Hines and Brown,[4] the vital capacity, an electrocardiogram, and x-ray film of the heart, were also obtained in most cases.

The results of the exercise tolerance test were not conclusive for 13 of the 100 cases, because in 12, the number of ascents on the steps was not standard, although the blood pressure and pulse were within normal limits at the end of two minutes and in one, the exercise was standard, but the pulse rate was high, due to many premature contractions. It was not possible to do more than one test on any of these subjects.

Two patients discontinued the exercise because of severe breathlessness although their blood pressure and pulse rate were not elevated. One of these had a tracheotomy, which presumably reduced his ventilation, and the other had a possible pericardial effusion which was never established as the true diagnosis.

Four discontinued the exercise because of weakness and giddiness after doing from 63 to 87 per cent of the standard exercise. In all cases the blood pressures and pulse rates were normal.

When patients complained of severe symptoms, they were usually allowed to do the first test at a rate that was comfortable for them, and as a result, the exercise was not standard in 15 other cases. In this latter group, however, there was evidence of diminished tolerance in the blood pressure or pulse rate, in spite of the substandard exercise.

CONSTANCY OF RESULTS

The exercise test was performed two or more times on 30 patients, and in 19 there was agreement in the tests performed on separate days; that is, the blood pressure and pulse rate were either within ten points of the resting level on both days, or exceeded ten points on both days; in ten cases they were within normal limits on both tests, and in nine cases they were interpreted as showing diminished tolerance in both tests. In the latter group, one patient with hypertensive heart disease showed dimin-

ished tolerance in three tests and after hospitalization for several weeks had a normal tolerance, although the blood pressure continued high. Two patients were examined a year after the first test and their reactions were practically identical in both tests.

In four patients, the systolic blood pressure remained elevated at the end of two minutes in the first test, but on the second examination the blood pressure and pulse rate were normal. One patient had mild hypertensive heart disease and the second examination was made four days later when he stated that he felt better. In one other instance, the examinations were six days apart, while the other two were examined on successive days and their reactions on the first day were only slightly below normal. These four patients were listed among those with normal exercise tolerance for the general tabulation. It is impossible to say what part hospital rest and relief of emotional tension played in changing the response; however, they are likely factors, as Master[3] reports that variation in the general well-being due to loss of sleep, excessive smoking, and various drugs such as the barbiturates, caused a reduction of tolerance in his normal series. Three of these four patients were given diagnoses of neurocirculatory asthenia.

In the remaining seven patients the two examinations were not comparable for various reasons. The exercise was not the same on both examinations in three cases; the blood pressure response being normal when less exertion was performed and excessive when more exertion was done; and obvious psychic disturbance in the other four patients prevented the development of proper basic conditions before or after the exercise in at least one of the tests.

It is apparent that uncontrollable factors, such as emotional instability, may cause blood pressure and pulse rate changes, indicating a decreased tolerance, which cannot be confirmed by other tests or other clinical methods. Although the results of this method of testing exercise tolerance were consistent in the majority of cases, the inconsistent results cast doubt on the interpretation of responses indicating decreased tolerance.

TYPES OF REACTION

Master[3] and Nylin[5] refer to the reports in the literature of observations showing the tendency for the systolic pressure to be elevated after exercise, while the diastolic pressure and pulse rate exhibit little variation.

The usual reaction in the 132 tests in our series was a moderate elevation of the systolic blood pressure above the resting level, two minutes after completing the exercise, with slight changes in the diastolic pressure and pulse rate above or below the resting level. In 52 instances the systolic pressure was more than 10 mm. of Hg above the resting level, which corresponded to similar excessive rises in diastolic pressure in only four cases and pulse rate increases of more than 10 beats per mm.

in twelve patients. Only three patients exhibited a significant increase of pulse rate who did not show a comparable elevation of the systolic pressure.

A considerable proportion of all the patients showed only a decrease in the diastolic pressure after exercise, perhaps because of failure to get a basic level during rest, while the systolic pressure was below the resting level two minutes after exercise in only 17 tests. Three patients exhibited a decrease of systolic pressure of more than 10 mm. of Hg. This could not be explained on the basis of observed changes in the cardiac mechanism, although they all presented marked circulatory symptoms and particularly asthenia. It was not possible to reproduce similar symptoms by stimulation of the vagus through the carotid sinus reflex and it was inferred that a peripheral mechanism was responsible for the failure to maintain the level of systolic pressure in these patients.

The pulse rate, counted by manual palpation of the radial artery, was below the resting level in approximately 25 per cent of all the tests. The decrease was more than 10 beats per minute in only three patients, two of whom had resting levels above 100 beats per minute. Bierring, Larsen, and Nielsen[6] state that they have never seen the pulse curve, after exercise, fall below the resting pulse in normal persons, and have seen it only infrequently in cardiac patients. These authors recorded the heart rate electrocardiographically, while in our series the decrease in pulse rate, after exercise, was observed by a less accurate method, and no particular significance was attached to it.

RELATION TO THE PRESSOR REFLEX

Since the systolic elevation of the blood pressure was the principal criterion for determining diminution of the exercise tolerance by the Master test, as indicated in the previous section, it was desirable to examine the reaction of the blood pressure to other stimuli than exercise.

Hines and Brown[4] proposed the cold pressor test which, they believe, indicates "the potential reactability of the vasomotor nervous system and that hyperreactions are synonymous with potential or existent hypertension, whether the levels of the blood pressure are increased or not." Assuming this to be true, a hyperreaction which is defined as a minimal increase in the systolic blood pressure of 26 mm. of Hg, or of the diastolic pressure of 22 mm. of Hg might also be reflected in the blood pressure reaction to exercise. The number of patients with hypertension in any group will affect the number showing a hyperreaction to cold, as a high percentage of patients with elevated blood pressure give an excessive reaction to the cold stimulus, according to Hines and Brown. This was demonstrated when the patients were classified according to the presence or absence of definite evidence of heart disease or hypertension. The heart disease group contained 61 per cent of hyperreac-

tors to the cold test, while the group without definite heart disease or hypertension contained only 39 per cent hyperreactors. Dividing the patients on the basis of their response to the step test, patients with hypertension appeared in both the normal and diminished groups but principally in the latter. The percentage of patients showing hyperreaction to the cold test was 35 per cent in the normal exercise tolerance group and 55 per cent in the diminished tolerance group.

In order to avoid an arbitrary division into groups, 83 patients were chosen who had completed both the pressor reflex and exercise tolerance tests on the same day. A correlation was run between the maximum systolic blood pressure elevation during the cold stimulus, and the elevation of the systolic blood pressure two minutes after exercise. The coefficient of the correlation derived was +9 out of a possible 100 for a perfect correlation, which expresses a very low grade relationship. On the basis of this comparison, the blood pressure and pulse rate responses to exercise were not apparently related in any important degree to the "potential reactability of the vasomotor nervous system" as shown by the cold pressor test, and might, with more assurance, be considered an evidence of response to factors outside the vasomotor system.

RELATION TO THE LILJESTRAND-ZANDER PRODUCT

Nylin[5] refers to the reports of Liljestrand and Zander who observed a linear relationship between the product of the "reduced amplitude" (ratio of pulse pressure over mean blood pressure) multiplied by the pulse rate, and the minute volume of the heart. In Nylin's report he showed that the Liljestrand-Zander product was retarded in its return to normal in heart patients with decompensation although the values for normal and decompensated individuals overlapped.

We calculated the Liljestrand-Zander product from our data for the blood pressures and pulse rates at rest and two minutes after completing the exercise, expressing the difference as a plus or minus percentage of the product of the values at rest. In our series, the average difference was +16.8 per cent (standard deviation 17.20) in the patients with normal tolerance, according to the step test, and +35 per cent (standard deviation 22.88) in the patients with diminished tolerances. The range of the percentage differences between the products at rest and after exercise was from +57 per cent to –13 per cent in the normal tolerance group and from +82 per cent to –19 per cent in the diminished tolerance group. Because of the large standard deviations, the differences in the two groups were not significant and would not indicate any correlation between the interpretation of the step test and the Liljestrand-Zander product. Our observations were not sufficiently prolonged to determine the time required for the product to return to normal values.

If the Liljestrand-Zander product has a linear relationship to the minute output of the heart, our data would indicate that the minute volume might be increased or decreased after exercise in both normal individuals and patients with heart disease, with a tendency for a greater increase in the latter group. Similarly, the values for the actual product varied widely and were often highest for those who presented the most evidence of heart disease. Starr, Collins, and Wood,[7] reported "certain patients with advanced myocardial disease have larger cardiac outputs than some normal persons" and concluded "these values throw very little light on the condition of the heart." Harrison[8] summarized the results of various investigations of the cardiac output in heart failure, stating "no parallelism exists between the severity of the symptoms and the level of the cardiac output."

Assuming, therefore, that the Liljestrand-Zander product does have a close relationship to the minute volume of the heart, the absence of significant correlation between the interpretation of the step test and either the product or the percentile change of the product after exercise, does not necessarily invalidate the step test as a method of determining functional capacity of the heart.

RELATION TO VITAL CAPACITY

The vital capacity was determined for 81 of the 87 patients for whom there was an exercise tolerance test that could be interpreted, and this was compared with the calculated theoretical capacity, based upon 2,500 c.c. per square meter of body surface as proposed by West[9] for normal individuals. It is well recognized that many factors besides pulmonary congestion due to heart disease will affect the vital capacity and that there is considerable variation among normal subjects, although the vital capacity for the same normal individual is quite constant.

The vital capacity of the patients with definite heart disease without any signs of failure varied from 50 per cent to 101 per cent of their calculated normals, while the vital capacity in the group without definite evidence of heart disease ranged from 55 per cent to 112 per cent of the calculated normal; the average for the two groups was 76 per cent and 83 per cent, respectively.

When compared with the results of the step test, regardless of the presence or absence of signs of heart disease, the patient with a normal tolerance had an average vital capacity of 82.5 per cent of the calculated normal and those with a diminished tolerance, an average of 78.3 per cent. Statistically this was not a significant difference. A frequency curve showed the peak of incidence at about 70 per cent of the theoretical normal capacity for both groups; the group with diminished tolerance had more cases below 60 per cent, while the group with normal tolerance had a greater number with 100 per cent or more of the calculated vital capacity.

In 20 patients the vital capacity was measured before and immediately after exercise. In all but one case, the latter reading was less than the first, the average reduction being 368 c.c. or about 10 per cent of the resting capacity. Levine and Wilson[10] reported only a 2 per cent reduction in normals exercised to breathlessness. As a rule the vital capacity was reduced less than 10 per cent and in some in which respiratory distress seemed most prominent, the percentage reduction was the least; thus it did not seem possible to correlate observed breathlessness with reduction in the vital capacity. Levine and Wilson[10] reported in patients with "irritable heart" that the breathlessness was out of proportion to the vital capacity, while the difference between mild and severe cases was not great. Arnett and De Orsay[11] and others have pointed out the variability of the vital capacity, and emphasized that the value of the examination is largely in comparing measurements, from time to time, on the same individual.

In our series, there was very little correlation between the vital capacity and definite signs of heart disease. On the basis of West's formula for normal men, Arnett[12] concludes that an absolute reduction of 1,644 c.c. from the calculated theoretical capacity is significant, being three times the standard deviation. Only 18 of our patients showed this amount of absolute reduction, some only after exercise, while several patients with severe heart disease, including two who died within a year with heart failure, did not show this much reduction. It appeared, therefore, that, as an isolated examination, the vital capacity did not correspond closely either to the tolerance to exercise, as estimated from the history and the step test, or to the evidence of heart disease in patients without definite failure.

RELATION TO DIAGNOSIS

The final consideration, in determining the usefulness of the two-step test for estimating exercise tolerance, was to compare the interpretation of the test with other clinical manifestations. For this purpose the patients were classified in four groups on the basis of their response to the step test and the presence or absence of definite evidence of heart disease. The groups were as follows: (1) diminished exercise tolerance without heart disease; (2) diminished exercise tolerance with heart disease; (3) normal exercise tolerance without heart disease; (4) normal exercise tolerance with heart disease. The first and last classifications presented the principal inconsistencies between the clinical signs and the results of the step test and will be discussed in most detail. It should be noted that, on the basis of history, practically all of these patients could be said to have had some impairment of functional capacity, although in many instances the physical examination did not reveal sufficient cause, either in the heart or other systems, to account for the disability.

In the group with diminished tolerance and without definite evidence of organic disease of the heart, there were 19 men with an average age of forty-five years, ranging from thirty-eight to fifty-seven years. All of these patients complained of symptoms suggesting circulatory dysfunction, and some had been told that they had heart disease. Breathlessness, on exertion, was one of the most common complaints, but fatigability and precordial pain were more often causes for disability. In several cases the principal problem was to determine whether thoracic pain was significant of the anginal syndrome. Five patients gave a history of repeated attacks of unconsciousness and two had suffered from convulsive seizures, of unknown cause, for many years. Several were chronic alcoholics; one had had a recent attack of acute cholecystitis; one had severe diabetes which was not properly stabilized; and one had had a recent postoperative respiratory complication. In addition to these illnesses, two patients presented the manifestations characteristic of severe neurocirculatory asthenia, and an associated "nervousness" or other psychoneurotic complaints were the rule rather than the exception throughout the group. At least nine of the 19 patients claimed that the onset of their symptoms occurred during their military service, about eighteen years previously, and most of these men had done very little work, because of their symptoms, since discharge from the service.

None of the patients in this group exhibited enlargement of the heart on physical examination and the teleroentgenograms showed no unusual configurations. The blood pressure was not consistently elevated in any of the patients; the highest systolic pressure was 158 mm. of Hg and the highest diastolic pressure was 90 mm. of Hg. There was no evidence of involvement of the heart valves in any patient.

Electrocardiograms were obtained for all patients in this group and were normal, with five exceptions. In one tracing, the voltage of the QRS complexes was 0.5 millivolt or less in all three leads, in another the heart rate exceeded 100 beats per minute, and in a third tracing the voltage of the T-waves in Lead I was less than 0.1 millivolt. An electrocardiogram was obtained on one patient, shortly after a fainting attack, which showed slight depression of the R-ST intervals, as compared with previous tracings. In the fifth patient a record was obtained during an attack of paroxysmal auricular fibrillation; however, on the following day after the cessation of the attack, an electrocardiogram was obtained which showed no abnormality. Changes, of the type enumerated above, have been described as occurring in patients with no apparent heart disease.

It could not be definitely determined whether some of the patients in this group had coronary arteriosclerosis with myocardial impairment, and whether the patient with paroxysmal auricular fibrillation had organic heart disease, but it was the clinical opinion, based on other

methods of examination, that the symptoms which were presented were not due to organic heart disease. If this was true, it would appear that the blood pressure and pulse rate responses to exercise may be exaggerated in the absence of organic disease of the heart when there is disease or dysfunction in other systems. Less than a year has elapsed since most of these patients were examined by the two-step test and subsequent events may indicate that greater specificity for the test might have been assumed.

There were also in our series, 15 patients who had definite signs of heart disease or hypertension who gave a normal response to the step test. The patients with resting blood pressures above 150 systolic and 90 diastolic were included in this group, whether cardiac enlargement or myocardial damage was demonstrable or not, although only two of the nine hypertensive patients showed any suggestive changes in the electrocardiograms. Of the remaining six patients, two had rheumatic heart disease, with mitral stenosis; one had congenital patent ductus arteriosus; and one showed a short auriculoventricular conduction time, with prolonged intraventricular conduction time, and a history of paroxysmal tachycardia. In this patient the lesion might have been a congenital abnormality and not necessarily disabling, except during the paroxysms of tachycardia. One patient was considered to have arteriosclerotic heart disease largely on the basis of a history of an acute coronary occlusion two years previously, although a small downward deflection (Q) in Lead IV was the only residual evidence of myocardial damage. Another patient was given a diagnosis of probable arteriosclerotic heart disease, on the basis of a history of pain on effort and low voltage T-waves in all leads. This patient's threshold for pain production must have been high because he did not develop pain while completing a test with twice the number of ascents standard for his age and weight.

It was conspicuous that the cardiac involvement in these patients when present was not severe and was not incompatible with a good exercise tolerance, indicating good myocardial reserve. They would have been grouped clinically in Classes I and IIA according to the American Heart Association rating of functional capacity.

Twenty-one patients were considered to have both a diminished exercise tolerance and heart disease, although in three patients, with the exception of slight hypertension, there was very little on which to base a diagnosis of heart disease. In general, the cardiac involvement exhibited was definite and was more severe in these patients than in those in the previous group. The electrocardiographic changes in 11 instances indicated myocardial damage, the average age was slightly higher (forty-eight years) and more patients were considered to have arteriosclerotic heart disease; but in some instances the cardiac reserve, estimated clinically from the history, was not less than in patients in which the step

test was within normal limits. At the time of the examination none of the patients had any signs of congestive failure, although one has subsequently progressed to this state, and one died following surgical treatment of a carcinoma of the rectum.

The largest group contained 32 patients who showed a normal response to the step test and had no definite evidence of heart disease. Two of these patients showed occasional premature contractions in the electrocardiogram; the voltage of the QRS complexes was low in two cases; two showed sinus tachycardia; and one a deep Q_3. In the absence of other changes it was considered that these deviations were not evidences of myocardial change. As in the other groups some of these patients complained of fatigue, dyspnea, palpitation, fainting, and pain in the chest. The exercise test was most in accord with the clinical findings in this group, as it supported the opinion that no organic disease of the heart was present when objective signs were not discovered upon which to base a diagnosis. Most of these patients appeared to belong to the psychoneurotic classification and diagnoses of neurocirculatory asthenia had been carried, in some instances, for many years.

HEART PAIN

Several of the patients in this series complained of precordial pain, which was not usually typical of significant heart pain, although the differentiation, entirely on history, was not satisfactory. These patients were given a standard test and if pain did not develop, they were instructed, at a subsequent examination, to make as many ascents as possible or until pain developed. In some cases such a procedure caused pain after a comparatively few ascents, but when the patient complained of general fatigue, without developing heart pain, the history of thoracic discomfort was largely discounted as evidence of serious coronary artery disease.

COMMENT

It is apparent from the preceding discussion that a decrease in the exercise tolerance, as manifested in the step test, was not considered to be an infallible evidence of heart disease, because various extracardiac factors, such as infections or abnormal mental states, with autonomic nervous system imbalance, might be responsible for the character of the blood pressure and pulse rate responses.

In this group of patients who complained of symptoms under slight to normal activity there was generally fair agreement between the results of the test, the extent of the cardiac involvement found on examination, and the estimation of the functional capacity from the history. In some instances, however, the diminished tolerance observed by the test was definitely inconsistent with the results obtained in other cases, judging

by the patients' statements as to their habitual activity and the degree of cardiac involvement. It does not seem warranted, therefore, to place much value upon this test as a dependable measure of decreased cardiac functional capacity.

Since the exercise was mild, it was not thought incongruous that some patients with organic disease of the heart should have a normal exercise tolerance by this test, indicating a fairly good cardiac reserve. Unless a maximum strain is imposed upon the heart, however, the earliest stages of heart failure cannot be differentiated, and the response of the patient with organic damage may be as good as that of the normal individual to milder exercise. For this reason normal exercise tolerance by the two-step test cannot safely be interpreted as indicating that no reduction in the functional capacity of the heart exists. On the other hand, no patient, who was judged clinically to have more than moderately reduced functional capacity, gave a normal response to the test.

With these limitations in mind, the test provides a means of examining a patient's response to exercise under reasonably standard conditions, but the interpretation of the test should not be given any decisive importance.

CONCLUSIONS

The test was easily performed and did not require complicated apparatus or much cooperation on the part of the patient.

The results were occasionally difficult to interpret and did not always agree on successive days when the same patient was tested.

The usual response, two minutes after completion of the exercise, was an elevation of the systolic blood pressure which appeared to be unrelated to the level of the resting blood pressure or the response to a cold water stimulus.

The relation of the sustained rise in the systolic blood pressure to the condition of the heart could not be directly demonstrated, and in the absence of definite organic disease of the heart, other conditions such as infections and psychoneuroses were associated with reactions characteristic of diminished exercise tolerance.

A diagnosis of heart disease is not warranted, based on the results of the two-step test alone.

A single test is not dependable evidence of reduced functional capacity and repeated tests that agree should be given only suggestive value.

The earliest stages of heart failure cannot be differentiated by the test, although normal responses are more dependable and indicate normal or only slightly reduced functional capacity.

A modification of the test is a useful method of studying patients who complain of thoracic pain.

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Department of Clinical Reports

TRANSIENT, RECURRENT, COMPLETE LEFT BUNDLE-BRANCH BLOCK

Report of a Case

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IN ALL probability transient, complete bundle-branch block is not a rare condition. Nevertheless, there have been very few recorded observations of this condition.

CASE REPORT

The patient, a man, aged sixty-eight years, first came to me on April 1, 1928, complaining of slight palpitation after playing eighteen holes of golf, and with this a feeling as though his heart were irregular. This was his chief complaint and only

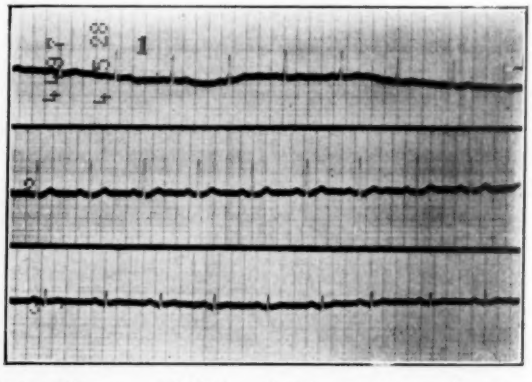

Fig. 1.—Electrocardiogram (4497)A. April 5, 1928. Slight palpitation after eighteen holes of golf. No evidence of intraventricular conduction. P-R, 0.16; QRS, 0.06.

occurred following prolonged exercise. Two years before I saw him he had had a bilateral herniorrhaphy, with an uneventful postoperative course. Remainder of past history and family history were noncontributory and irrelevant.

On physical examination he appeared a robust, well-preserved elderly man lying perfectly flat on the table without discomfort. He had a fairly marked coarse tremor of both hands and evidence of generalized arteriosclerosis. His heart was not enlarged, rate was moderate, with regular sinus rhythm; there was a slightly rough short systolic murmur at the base. The remainder of the physical examination was essentially negative. Likewise on fluoroscopy the heart presented an average silhouette, except for slight widening of the aortic arch shadow.

Laboratory Findings.—The blood Wassermann reaction was negative. Red blood cells numbered 5,100,000, and the hemoglobin was 108 per cent. The urine showed

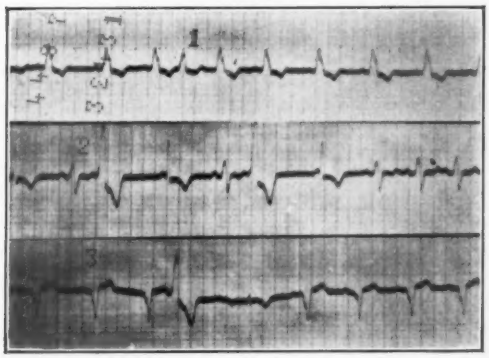

Fig. 2.—Electrocardiogram (4497)B. March 24, 1931. Patient having occasional attacks of palpitation. Complete left bundle-branch block. Numerous auricular and ventricular extrasystoles from different foci. P-R, 0.16; QRS, 0.16.

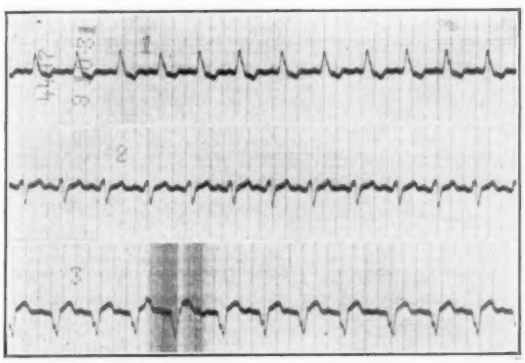

Fig. 3.—Electrocardiogram (4497)C. Sept. 10, 1931. Patient playing nine holes of golf twice a week. Asymptomatic. Complete left bundle-branch block. P-R, 0.16; QRS, 0.16.

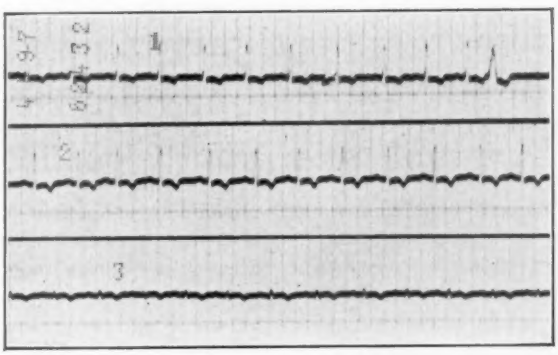

Fig. 4.—Electrocardiogram (4497)D. June 24, 1932. Asymptomatic. No conduction defect. T-waves negative in all three leads. One ventricular extrasystole. P-R, 0.16; QRS, 0.06.

a faint trace of albumin, which was not always present in subsequent specimens. Nonprotein nitrogen was fairly constant and was around 42 mg. per 100 c.c. throughout nine years of observation.

Clinical Course and Electrocardiograms.—Ten electrocardiograms were taken from first office visit in April, 1928, through the most recent examination in February, 1937. The first tracing (Fig. 1) showed no significant findings. The second (Fig. 2) in March, 1931, three years later, revealed a complete left bundle-branch block. Several weeks prior to this he had had an automobile accident without serious injury, but following this he had had palpitation frequently. In September, 1931, six months later, the bundle-branch block was still present (Fig. 3) and in addition, T_2 had changed from negative to positive. At this time he was relatively asymptomatic and

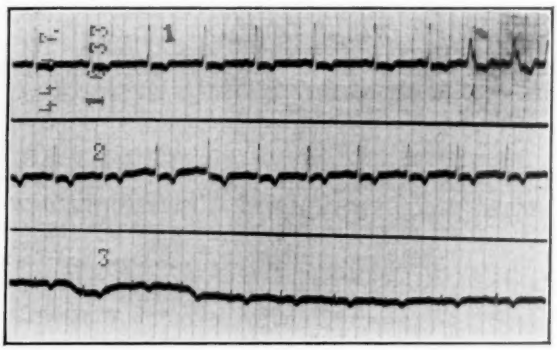

Fig. 5.—Electrocardiogram (4497)E. Jan. 6, 1933. Continues asymptomatic. Note last three complexes in Lead I. P-R, 0.16; QRS, 0.06. P-waves present. QRS, 0.16.

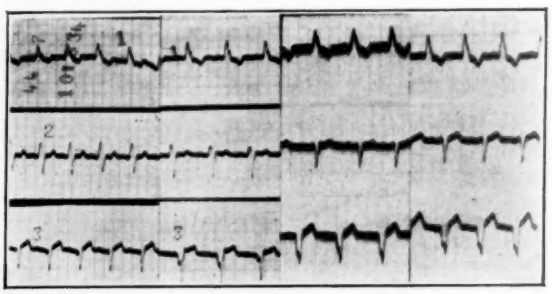

Fig. 6.—October, 1934(G). April, 1935(H). March,1936(I). February, 1937(J). Electrocardiogram (4497) October, 1934, to February, 1937. No cardiac symptoms. Permanent left bundle-branch block.

was playing nine holes of golf a day. When next seen on June 24, 1932, nine months later, his block had disappeared, and T_2 had changed back from positive to negative (Fig. 4). This was the first available evidence of the transient nature of this block, and at the time of his visit in January, 1933, eight months later, quite by accident an interesting demonstration of this was obtained. The electrocardiogram (Fig. 5) caught three complexes in Lead I, showing bundle-branch block, while the remainder of the tracing was normal, except for T-wave negativity in all three leads. Nine months later a left bundle-branch block was present in all three leads (Fig. 6), and this persisted throughout four subsequent tracings taken at approximately yearly intervals. Throughout this entire period the patient has con-

tinued to live an active life mentally and physically and, except for a little palpitation occasionally, a little dyspnea on moderately severe exertion, and recently a few dizzy spells, he has been symptom-free.

DISCUSSION

Conduction bundle-branch block defects are generally considered manifestations of severe myocardial damage, and when present usually remain as a fixed condition throughout life. Few exceptions to this observation have been recorded. This applies only to bundle-branch block as a manifestation of organic heart disease and does not include the so-called functional bundle-branch block described by Sigler,[1] or those cases of bundle-branch block with short P-R interval in healthy individuals recorded by Wolff, Parkinson and White,[2] and since described by many others. Nor does it include those transient episodes of impaired conduction of the bundle induced by the combined administration of digitalis and quinidine.

Attention has been called recently to the infrequency or recurrent bundle-branch block by Willius and Anderson,[3] who added one case to the six previously described in the literature; of these seven cases four were examples of complete and three of incomplete bundle-branch block. Willius and Anderson[3] point out that in all the six cases previously described in the literature the episodes of conduction bundle defect were associated with periods of cardiac insufficiency; in one they were related to attacks of paroxysmal auricular fibrillation; in the others they were associated either with attacks of pulmonary edema or with other manifestations of decompensation and disappeared when compensation was restored. The case of Willius and Anderson[3] was of particular interest because of the complete absence of cardiac symptoms at any time in spite of the complete bundle-branch block as well as prolonged A-V conduction-evidence of fairly profound interference in impulse conductivity.

SUMMARY

A case of transient recurrent complete left bundle-branch block followed over a period of nine years with electrocardiograms is reported, and the meager literature is briefly reviewed.

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DISSECTING ANEURYSM OF THE AORTA: ANTE-MORTEM DIAGNOSIS AND COURSE FOR FIFTY-THREE DAYS*

CASE REPORT

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THE clinical diagnosis of dissecting aneurysm of the aorta has been made with increasing frequency in the past two or three years. Yet the number of cases reported is few and the following typical case is

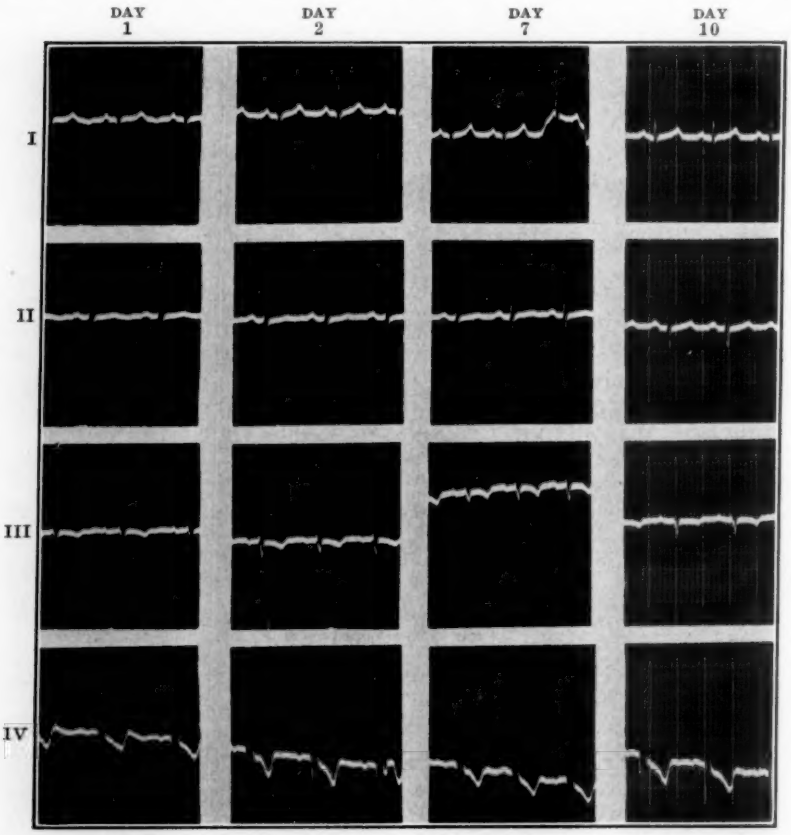

Fig. 1.—Serial electrocardiograms taken on the first, second, seventh and tenth days of illness. There is no evidence of coronary occlusion.

reported in an attempt to continue the interest that has been stimulated. The observations on this patient cover a period of fifty-three days.

CASE REPORT

The patient, T.S., was a forty-five-year-old colored male janitor admitted to Grady Hospital April 20, 1937, with the complaint of severe abdominal pain.

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Past History.—His past history contained few important items. Nine years previously he had been treated for an inguinal adenitis associated with recurrent gonorrhea. No Wassermann test of the blood was reported and no estimation of his blood pressure was made.

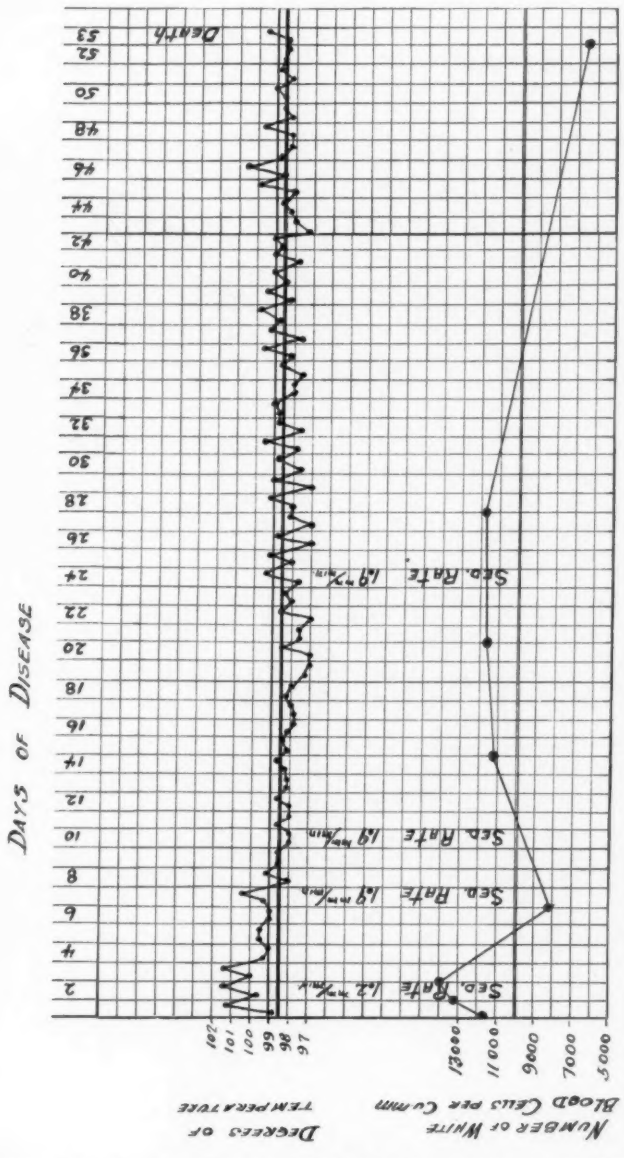

Fig. 2.—Temperature chart and variations in the white cell count and sedimentation rate of the blood during the patient's illness.

Three years prior to his present admission he had a penile sore diagnosed as primary syphilis. He stated that a Wassermann test on his blood was positive at that time and that he received 16 injections for syphilis. During and since that time the patient had worked regularly as a janitor and had had no complaints.

Present Illness.—His present illness began at 1:00 A.M. on April 20, 1937, at which time he was awakened with a severe pain under the lower angle of the left scapula. The pain was sharp and constant and at first did not radiate. It was not described

as "tearing" by the patient. The pain was so severe that he was taken to the emergency clinic where he was given one-fourth of a grain of morphine and returned home. His pain was not lessened. About two hours after the onset, the pain began to move around to the left flank and to the abdomen about the navel. It remained constant and excruciating in character. There was some nausea but no vomiting. The patient had "cold sweats," but he did not have a "fear of impending death." Seven or eight hours after the pain began it became slightly less severe and at this time the patient was seen in the medical clinic.

Examination.—He was a muscular colored man, obviously suffering from pain. His face was drawn and he groaned considerably. He was more comfortable bending slightly forward than in other positions. There was engorgement of the neck veins, reaching on the left to the lobule of the ear and on the right only one-fourth of this

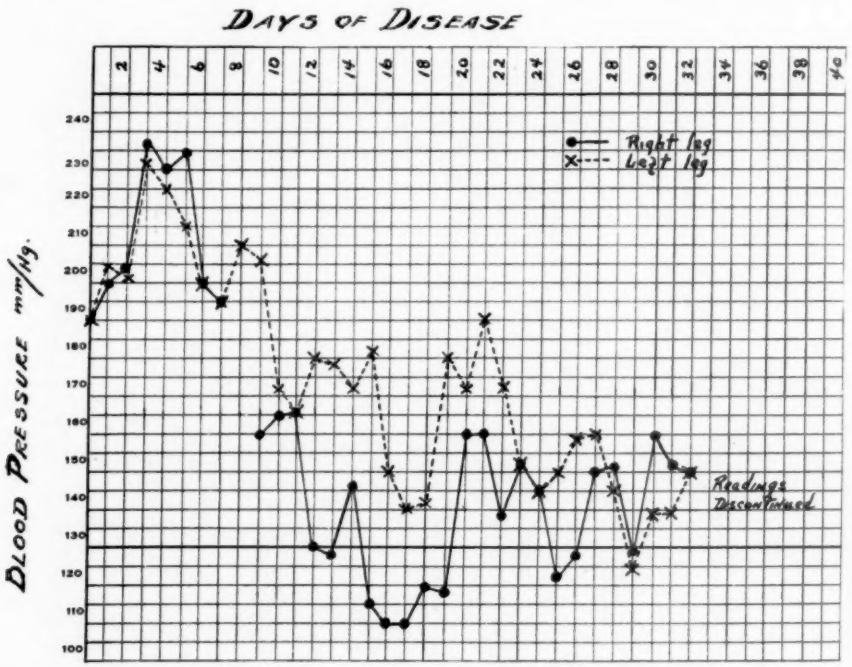

Fig. 3.—Comparison of the systolic blood pressure in the right leg and in the left leg during the first thirty-two days of patient's illness.

distance. The patient's temperature was 98.4° F., his respirations numbered 30 per minute, his pulse 80 beats per minute. The pupils of his eyes were equal on the two sides. On examination of the ocular fundi, a well-advanced sclerosis of the arteries was found; there were no hemorrhages. Examination of the lungs revealed no abnormal findings. There were no abnormal pulsations about the neck and chest. The radial and brachial arteries were sclerotic and tortuous, and the pulse of the two arms was synchronous and of good excursion. The apex beat of the heart was visible in the fifth interspace and was forceful and even. The area of cardiac dullness was normal in size. The sounds of the heart were of fair quality, there was no gallop rhythm and there were no murmurs. The aortic second sound was accentuated. Estimations of the blood pressure in the four extremities were as follows: Right arm 240/140 mm. Hg, left arm 240/140, mm. Hg, right leg 270/160 mm. Hg, left leg 270/160 mm. Hg. The abdominal examination showed nothing significant, the descend-

ing aorta could be felt pulsating. The white cells in the blood numbered 7,000 per cubic millimeter. A probable diagnosis of dissecting aneurysm of the aorta was made. This opinion was further supported by fluoroscopic examination of the heart a few hours later, which showed a diffuse enlargement of the aorta below the arch, with pulsations of it anteriorly and not posteriorly. Also during this first day further evidence of a dissecting aneurysm was found by a demonstration of a difference in the venous pressure in the two arms, measuring 110 mm. water in the right arm, and 350 mm. water in the left arm.

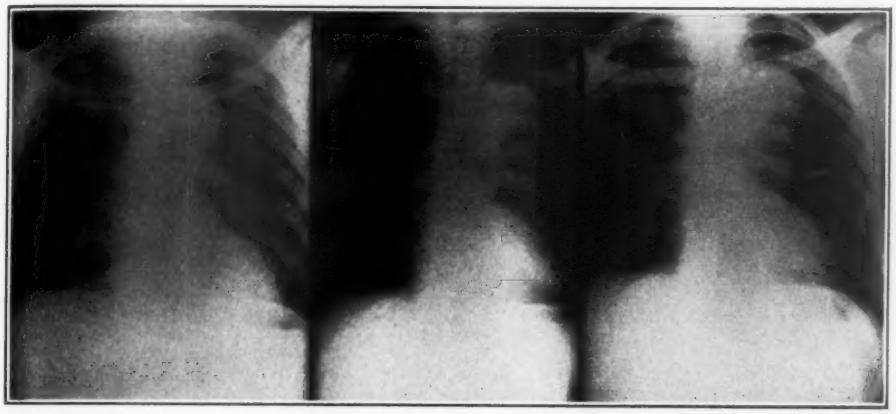

Fig. 4.—Roentgenograms taken in anteroposterior position on the first, thirty-first, and forty-third days of illness.

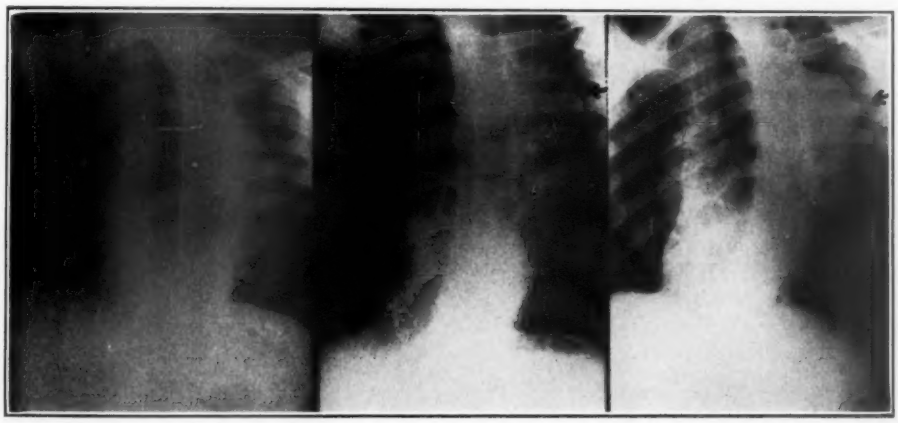

Fig. 5.—Roentgenograms taken in the right oblique position on first, thirty-first, and forty-third days of illness. Note the local bulge where the final rupture occurred.

Course.—The patient remained in the hospital for fifty-three days prior to death and further observations of interest are presented in Figs. 1 to 7. The Wassermann reaction of the blood was negative on three occasions. In the first part of the patient's illness albumin was present in the urine and later it was absent. Serial electrocardiograms (Fig. 1) taken on the first, second, seventh and tenth days showed no evidence of coronary occlusion. In Fig. 2 is a record of the variations in temperature, white blood cell count, and sedimentation rate of the blood. The number of white cells in the blood was usually elevated but was within normal limits on the day before death of the patient. The sedimentation rate of the blood was ab-

normally rapid on four estimations. In Fig. 3 is shown the daily variation in the systolic blood pressure in the two legs. The diastolic blood pressure remained parallel to the systolic. On the seventh day pulsations in the right femoral artery ceased for two days, but returned later; however, the blood pressure in the right leg remained at a lower level than that in the left leg for a period of about six weeks. The cause of this occlusion is shown in Fig. 7 to be a large thrombus surrounding the right iliac artery. In Figs. 4 and 5 are shown roentgen ray examinations of the heart on the first, thirty-first, and forty-third days, in anteroposterior and right oblique views. In the oblique view a

Fig. 6.—Photograph of the heart and upper aorta. The intimal tear has been cut into and appears just above (*a*); behind it may be seen the deep sac and the opening of the external rupture.

saccular outpouching of the adventitia can be seen in the aneurysm and the pointer shows about where final rupture occurred into the left pleural cavity on the fifty-third day.

During the first week of his illness the patient suffered with pain to the extent that morphine was required frequently to give him comfort. When the occlusion of the right iliac artery occurred (seventh day) he had a mild pain and numbness in the right leg and it became cool to the touch. This lasted only two days and ceased coincident with the gradual return of pulsations in the right femoral artery. Often during his illness the patient would have a moderate exacerbation of the abdominal

pain, but it was never as severe as the original pain. Changes in the physical examination were scarce. In the third week an area of dullness was noted in the upper dorsal chest and the area gradually increased in size during the weeks prior to death. The patient improved subjectively during his hospitalization and on the fifty-second day and fifty-third day he was allowed to sit in a chair. He died suddenly and quietly during the night in bed, fifty-three days after the onset of his disease.

Post-Mortem Examination.—Necropsy showed death to have been caused by rupture of the aneurysm into the left pleural cavity. The pleural space contained about three liters of blood. The final rupture of the aorta occurred in the outpouching

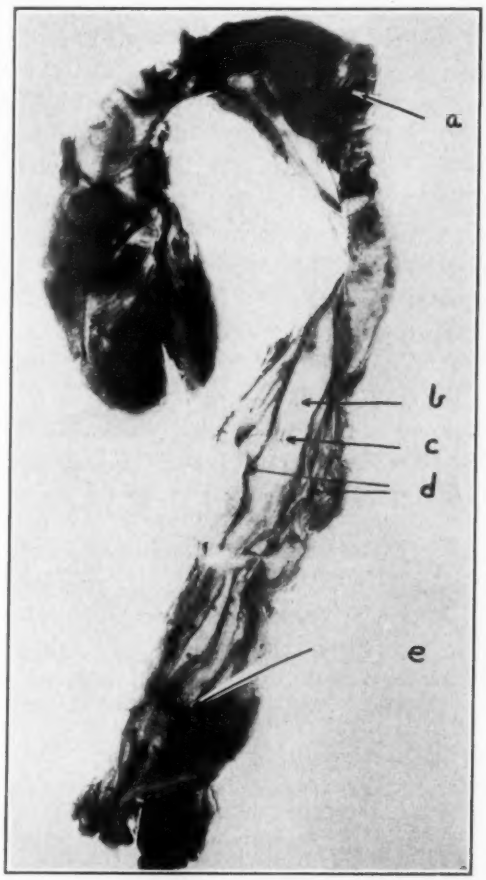

Fig. 7.—Photograph of the heart and entire aorta. (*a*) Site of rupture into left pleural cavity. (*b*) Orifice of celiac axis from which the vessel was torn by the dissecting column of blood. (*c*) Orifice of superior mesenteric artery. (*d*) Renal arteries; they were not torn loose. (*e*) Pointer in the orifice of the right iliac artery and below the pointer may be seen a large laminated blood clot.

shown in Fig. 5. This sac contained a laminated thrombus adjacent to the point of rupture, the tear measuring 2 by 0.25 cm. It was located in the anterior wall of the aorta, was diagonally situated and was 16 cm. distal to the aortic valve (Figs. 6 and 7). The dissecting column of blood had torn away several intercostal arteries and also the celiac axis and superior mesenteric artery (Fig. 7). However, no change due to deficient blood supply was visible in the intestines because the lumens of these large vessels were patent into the external column of blood and no thrombi were

present. The dissection of the aortic wall continued downward to and around both iliac arteries and a large laminated clot (6 by 5 cm.) surrounded the right iliac artery (Fig. 7).

The heart was not enlarged, weight was approximately 350 gm. The coronary arteries were patent and only slightly sclerosed. The aorta showed no evidence of syphilis grossly or microscopically and showed moderate atheromatous changes. Microscopic sections of the aorta showed that the dissection took place between the middle and outer thirds of the media, the usual location.

COMMENTS

In the past six months three thorough reviews,[4, 5, 6] of this subject have been published. Roesler and his coworkers have accepted 17 cases of dissecting aneurysm of the aorta diagnosed ante mortem and proved by autopsy. Seven other cases should be added to this number; another case reported by White and his associates;[4] three recently reported by Paullin,[6] one reported by Blackford and Smith,[7] one diagnosed by Vaughan and reported by Samson,[1] and one reported by Gurin and his coworkers,[8] diagnosed and proved at operation. These with the case reported bring the total number to 25 reported cases correctly diagnosed ante mortem and proved.

SUMMARY

A case of dissecting aneurysm of the descending aorta is reported in which the diagnosis was correctly made and in which observations on the patient were made during fifty-three days prior to death.

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SPONTANEOUS RUPTURE OF THE AORTA WITH HEMOPERICARDIUM CAUSED BY COARCTATION

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COARCTATION or stenosis of the aorta (adult type) can be described as a congenital maldevelopment of the descending arch of the aorta either at or just below the insertion of the ductus arteriosus. The coarctation may vary in degree, from a moderate narrowing of the great blood vessel to an abrupt constriction and, in some cases, complete obliteration. Clinically, coarctation of the aorta is marked by the development of a collateral circulation the extent of which is dependent on the severity of the stenosis, with forceful pulsation in the episternal notch and carotids, retardation or absence in the femorals and tibials, and hypertension in the upper extremities in conjunction with a decreased blood pressure in the lower part of the body. Abbott[1] believes the presence of these signs alone justify a tentative diagnosis of stenosis. Other investigators have emphasized the value of roentgen examination and electrocardiographic study as confirmatory evidence. The x-ray film may show erosion or scalloping of the under surface of the ribs, depending upon the size and vigorous pulsations of the collateral vessels, hypertrophy of the heart particularly of the left side, dilatation of the ascending aorta, absence of the normal aortic knob and dilatation of the first part of the aortic arch. Fray[2] feels that it is possible to establish the diagnosis in almost every case where a complete x-ray study of the heart and aorta can be made. There have been a number of cases of coarctation reported in recent literature, the diagnosis based entirely on clinical and x-ray findings.[3-11]

The case to be presented is one of extreme stenosis (adult type) of the arch of the aorta with death caused by spontaneous rupture of the ascending portion and resulting hemopericardium. In analyzing the modes of death from coarctation, Abbott[1] found spontaneous rupture occurring in only 33 cases in a series of 200. There have been four cases reported since 1928.[12-15] Although the diagnosis of coarctation was not definitely made previous to autopsy, it was suspected as the possible causative factor of the massive pericardial effusion which obscured the heart signs.

CASE REPORT

C. McI., an unmarried white male of nineteen years, was admitted to the Delaware County Hospital on Oct. 13, 1935, complaining of pain in the upper portion of the chest, the lower part of the neck, and a "thumping" above the sternum, these symptoms having developed suddenly during his attendance at an evening outing in a park adjacent to the hospital. There was no history of weakness or disturbance

of circulation in the lower extremities. During high school, the patient was above the average in scholastic ability and his athletic activities included the winning of the school letter in basket ball and football. Following an attack of measles at the age of eight years, a physician apparently told the mother that the patient had "heart disease," but of minor importance. In 1934, during a routine physical examination for freshmen in college, it was discovered that he had high blood pressure which was again noted approximately ten months later when he attempted to obtain a position. His father died of heart disease; the mother was living and fairly well; one brother and one sister were living and well.

On Oct. 15, 1935, the writer was called to see the patient in consultation with Dr. H. Lennox H. Dick. Examination revealed a well-developed and well-nourished young man, sitting upright in bed, who was dyspneic, slightly jaundiced, and who appeared gravely ill. There was marked pulsation with a distinct thrill over the right side of the neck about one inch above the clavicle and a similar finding in the suprasternal notch. The upper limit of cardiac dullness was not determined as it seemed to extend into the neck. In the second interspace, dullness extended 4 cm. to the right of the midsternal line and 5 cm. to the left; in the fourth interspace, 3 cm. to the right; left border 14 cm. to the left where the apex beat was distinctly felt. Point of maximal impulse was located in the fifth interspace about 11 cm. from the midsternal line. The heart sounds were regular and rapid, with a loud, rough, systolic murmur over the body of the heart, the aortic area, and on the left side posteriorly beneath the scapula. The blood pressure at this time was 190/150 in the left arm and 130/90 in the right as compared to 194/129 on admission (arm not stated); blood pressure in the lower extremities was not attempted because of the grave condition of the patient. The pulse rate was 128 and the temperature 98. The lungs were clear. A diagnosis of large pericardial effusion or hemopericardium due to rupture (cause undetermined, possibly coarctation of the aorta) was made.

Laboratory examination showed the blood count to be average, except for a white count of 18,400 with 81 per cent neutrophiles; the blood Wassermann and Kahn were negative. Blood chemistry, nonfasting, showed sugar 117.5 mg., urea nitrogen 25.2 mg., and creatinine 1.6 mg. The urine was clear.

An x-ray film of the chest taken Oct. 14, 1935, by Dr. Paul Bishop, showed marked enlargement of the heart, the right side more than the left. The aorta was widened but there was nothing to suggest aneurysm. The lung fields were relatively clear except for some trunk shadow which was probably circulatory in origin. Two days later the chest x-ray was repeated with some increase in the size of the heart noted and a very definite increase in the vertical diameter of the left side of the heart with obliteration of the major portion of the concavity of the left border and heart vessels. This change in so short a time was in support of the diagnosis of pericardial effusion.

Electrocardiographic study of October 16, revealed a P-R interval of 0.10 second; there was a suggestion of a Q-wave in Lead I and a definitely positive Q-wave in Lead III. The T-wave was erect in Leads I and II, inverted in Lead III, and diphasic in Lead IV. There was a marked elevation of the RS-T segment in Leads I and II. The tracing was suggestive of marked myocardial damage and pericardial irritation.

Paracentesis of the pericardium was attempted on October 16 but no fluid was obtained and the patient's condition was not altered by the procedure. His pulse was rapid, the heart action and signs were the same as previously noted. On the following day, October 17, the general condition of the patient was unimproved; the apex beat was still located in the fifth interspace about 14 cm. from the midsternal line; the murmurs were present and there was a thrill on palpation over the sternocleidomastoid muscle. During the day, his blood pressure dropped to 130 systolic in the right arm and 150 systolic in the left. The patient expired suddenly at 9:45 P.M.

AUTOPSY REPORT BY DR. A. D. WALTZ

The body was that of a white male, nineteen years old, about six feet tall, well developed and of an athletic build. No difference could be detected between the development of the upper and lower extremities.

On opening the chest, the pericardial sac was found to extend from the left chest wall to a point about 3 cm. to the right of the sternum, and from the clavicle

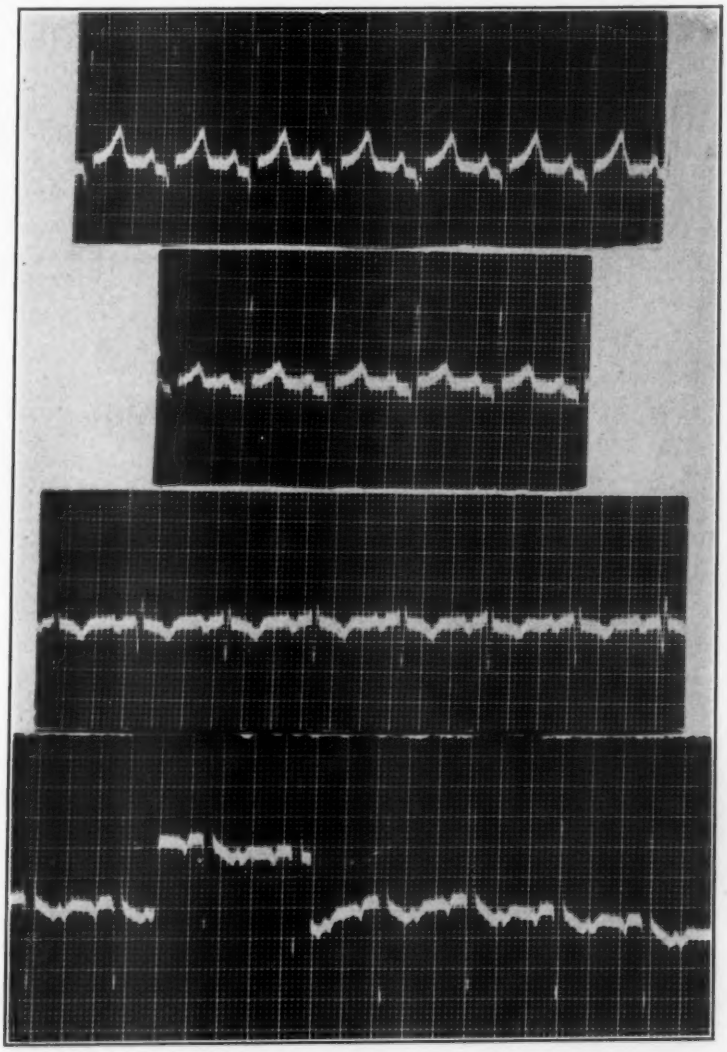

Fig. 1.—Electrocardiogram showing marked elevation of the RS-T segment in Leads I and II, particularly Lead I. Note the practically normal chest lead.

to the fifth intercostal space, measuring 21 × 19 × 10 cm. The pericardium contained a huge blood clot (495 gm.) surrounding the heart. The heart measured 14 × 12 × 9 cm. and with the pericardial sac weighed 770 gm. The total mass, heart and the blood clot within the pericardium, weighed 1,265 gm. The heart was removed with a portion of the descending aorta. The aortic arch was dilated to 4.8 cm. in diameter; the descending aorta was kinked and narrowed at a point 1 cm.

distal to the left subclavian artery. The lumen at this point was nearly occluded by a diaphragm-like obstruction with a central opening only 0.5 cm. in diameter. The aorta below this point was 1.8 cm. in diameter, definitely smaller than normal. The arch of the aorta showed a diagonal, zigzag tear in the posterior wall 2 cm. long, 1 cm. above the aortic valve. Blood had passed through this tear, burrowed between the adventitia and media of the aortic arch, and produced a dissecting aneurysm which had ruptured into the pericardial sac. The pericardium, the epicardium, the pericardial clot, and the edges of the aortic tear were roughened by early fibrous adhesions. The circumference of the valve orifices were: mitral

Fig. 2.—Arrow indicates the stenosis which has been increased in size during photography. *A* shows the outer wall of the dissecting aneurysm, mesial to which part of the blood clot can be seen.

valve 9.0 cm., aortic valve 7.5 cm., tricuspid valve 11.0 cm., pulmonary valve 8.0 cm. The thickness of the muscle walls measured: left ventricle 2.3 cm., left auricle 0.3 cm., right ventricle 0.9 cm., right auricle 0.2 cm. The valves were normal and the muscle was red and firm.

The coarctation of the aorta was not discovered until the heart had been removed, therefore no satisfactory study could be made of the collateral circulation to the lower extremities. However, there was no apparent enlargement of the intercostal arteries.

Other post-mortem findings were those secondary to heart failure; congestion and edema of the posterior portion of both lungs, congestion of liver, spleen, kidneys,

bladder and gastrointestinal tract. The anterior portion of the left lung was partially compressed by the large pericardial mass.

SUMMARY

A case with autopsy is recorded of congenital stenosis (adult type) or coarctation of the arch of the aorta in a white, unmarried male of nineteen years, with death caused by spontaneous rupture of the ascending portion of the great blood vessel and resulting hemopericardium. This patient had suffered neither cardiac embarrassment nor disability of any kind previous to the sudden fatal attack. Electrocardiographic study revealed pericardial irritation and confirmed the diagnosis of hemopericardium. The autopsy showed (1) a greatly hypertrophied heart which, together with the blood clot within the pericardium weighed 1,265 gm., the clot alone being 495 gm.; (2) dilatation of the aortic arch and a constriction of the descending portion 1 cm. distal to the left subclavian artery, at which point the lumen was nearly occluded by a diaphragm-like obstruction with a central opening only 0.5 gm. in diameter; (3) a diagonal tear 2 cm. in length in the posterior wall of the ascending aorta, 1 cm. above the aortic valve through which blood had passed producing a dissecting aneurysm which had ruptured into the pericardial sac. The valves were normal and there was no evidence of any infective process. Other postmortem findings were those secondary to heart failure.

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Department of Reviews and Abstracts

Selected Abstracts

Møller, K. O.: The Effect of Cocaine and Procaine on the Action of Adrenalin on Skin Vessels: Vascular Action of Cocaine and Procaine in the Perfused Rabbit's Ear. Arch. Internat. de pharmacodyn. et de thérap. **57**: 51, 1937.

Rabbits' ears were perfused according to the method of Katz, in which the change in pressure required to maintain the flow of fluid through the organ constant is used as an index of change in caliber of the small vessels. The author confirms the fact that perfusion with cocaine (1/1 million in Tryode) augments the constrictor action of adrenalin and finds that procaine does not. Cocaine itself constricts the vessels, but its action in sensitizing the vessels to adrenalin cannot be considered synergistic, since if adrenalin is added first, it does not increase the action of cocaine. The constrictor action of racemic corbasil (3:4-dioxy-nor-ephedrine) is also enhanced by cocaine.

STEELE.

Møller, K. O.: Contributions to the Pharmacology of Corbasil (3:4-Dioxy-nor-ephedrine) With an Analysis of the Effect of Cocaine and Procaine on the Vascular Actions of Adrenalin and Corbasil. Arch. Internat. de pharmacodyn. et de thérap. **57**: 67, 1937.

The author points out that corbasil and adrenalin are optical isomers, and that corbasil is also a vasoconstrictor but a considerably weaker one than adrenalin. The methods used for demonstrating the vasomotor effects were (1) in the extremities, recording of systemic arterial pressure and of pressure in the peripheral stump of the femoral artery maintained by collaterals and (2) in the viscera, the recording of systemic arterial pressure and volume of the organ studied. The author reaches the following conclusions:

1. Fundamental differences exist between the action of adrenalin and its isomer corbasil. Corbasil usually dilates the renal vessels, adrenalin contracts them; corbasil increases the volume of the spleen, adrenalin reduces it. In cats under ether or urethane anesthesia, adrenalin produces a fall in arterial pressure, corbasil a rise.

2. The ratio of action of adrenalin to corbasil differs in different animals.

3. Cocaine increases the pressor action of corbasil as well as that of adrenalin, and this increase is believed by the author to be due partly to the action of cocaine upon the peripheral vessels and to its inhibiting action upon the pressure regulating reflexes in the vasosensory zone.

4. Procaine sensitizes the organism to adrenalin and corbasil to a much lower degree than does cocaine, and the sensitization is supposed to depend exclusively upon inhibition of the blood pressure regulating reflexes originating in the vasosensory zone.

STEELE.

Rein, Hermann: The Physiologic Basis for the Mode of Action of the Experimental Substance "Knoll H 75" (1:p-Oxyphenyl 2. Methylamine-propane). Klin. Wchnschr. **16**: 700, 1937.

The effect of this drug (trade-mark Veritol) was studied in dogs under pernoctone-morphine anesthesia. Arterial pressure, pulmonary blood flow, venous pressure and the local circulation of the viscera or extremities were recorded optically, and often the flow of blood was recorded by Rein's stromuhr.

Respiration, rate of metabolism, and level of blood sugar were unaffected. The important action of the drug was found to occur when arterial pressure was low (operative shock, hemorrhage, etc.). Under these circumstances a sustained rise of pressure was easily obtained by intravenous injection of as little as 0.5 mg. If the arterial pressure was normal, in the intact animal only a very slight rise, if any, was observed. The inference is that the drug does not interfere with the ordinary reflex regulation of arterial pressure. The vagal type of pulse with slowing frequently occurred; but, if the vagi were sectioned or if the animal was atropinized, a rise of arterial pressure well above normal occurred even if it was at normal levels to begin with. The vagal influence was, in this way, shown to be important in preventing the rise of pressure following the use of the drug.

The site of action of the drug is believed to be different from that of adrenalin for two reasons: (1) the latent time of the reaction is 11 or 12 seconds instead of 7 or 8 seconds for adrenalin, and (2) in instances where adrenalin causes a fall veritol occasions a rise. Rein concludes that the drug may be very useful in operative or infectious circulatory collapse because of its marked activity and low toxicity.

STEELE.

Nielsen, H. E.: Influence of Body Position on the Cardiac Minute Output. Acta med. Scandinav. **90**: 456, 1936.

In the normal individual the cardiac minute output is increased 10 per cent and the stroke volume, 20 per cent, when the patient changes his position from sitting to lying down. In persons with heart disease the differences increase to 20 and 30 per cent, respectively. The respiratory ventilation with a similar change in position decreases 9 per cent.

KATZ.

Büssemaker, J.: The Influence of Restorative Drugs Upon the Orthostatic Circulatory Collapse at Diminished Environmental Pressure. Ztschr. f. d. ges. exper. Med. **100**: 808, 1937.

Normal men stood up in a pneumatic chamber at a pressure which corresponded to a height of 6,000 m. In from ten to thirty minutes the arterial pressure began to fall slowly at first, then more rapidly. The subjects exhibited faintness, heaviness of the limbs, air hunger, and dizziness. Just before complete collapse occurred, the drug was given intramuscularly. Drugs with central site of action (cardiazol, neospiran, coramine, cormed) restored arterial pressure promptly; those with peripheral action were less effective (sympathol, ephedrine). The author explains the greater usefulness of the centrally acting drugs by an increase in reflex excitability which yields an increase in tone throughout the peripheral circulation—arterioles, venules and capillaries—while the peripherally acting drugs affect only the arterioles.

STEELE.

Böhme, W.: The Action of Ventricular Systole in Accelerating Venous Flow. Klin. Wchnschr. **15**: 1631, 1936.

With the help of the roentgenkymograph and the indirect roentgencinematograph, there was found in man evidence pointing toward systolic acceleration of venous flow, as previously shown in animals. In high grade mitral and tricuspid insufficiency, the contour of the venous pulse is reversed. These studies emphasize the importance of the systolic suction of the ventricle in aiding venous flow.

KATZ.

Radnai, P., and Mosonyi, L.: The Significance of the Reflex Excitability of the Vagus in Angina Pectoris. Klin. Wchnschr. **16**: 228, 1937.

Pulse slowing was studied following eyeball pressure, pressure on the carotid sinus, and in the Valsalva experiment. It was found that these tests indicated increased vagus reflex excitability in patients with angina pectoris. Thus those patients with hypertension and anemia who also had angina pectoris had a higher vagus excitability than those without angina.

KATZ.

Scherf, D., and Schönbrunner, E.: The Pulmonocoronary Reflex in Lung Emboli. Klin. Wchnschr. **16**: 340, 1937.

This is a report of two cases with small pulmonary emboli in which marked electrocardiographic changes occurred without evidence of heart damage. This was considered to be due to a reflex coronary vasoconstriction from the lungs.

Electrocardiograms were taken on ten anesthetized dogs during production of small experimental pulmonary emboli. In 3 of these a typical Q_1-T_1 type of electrocardiographic change was found which is ascribed to reflex coronary vasoconstriction.

KATZ.

Ronchese, F.: Infra-Red Photography in the Diagnosis of Vascular Tumors. Am. J. Surg. **37**: 475, 1937.

Infra-red ray photography is a valuable method of investigation in the diagnosis of vascular cutaneous tumors in the living, when the presence of blood is doubtful.

AUTHOR.

Caeiro, Agustin, and Orias, Oscar: The Phonocardiogram Registered in Special Areas of Auscultation. Rev. argent. de cardiol. **4**: 71, 1937.

By optically recording the heart sounds (Wiggers and Dean) from the apex, mesocardiac and basal (pulmonic) areas simultaneously with the jugular pulse and the electrocardiogram in twenty healthy young medical students, the following conclusions were drawn:

An auricular sound was recorded in 85 per cent of the cases, with about 29 vibrations per second, lasting for about 0.10 sec., appearing in the records with an amplitude of 6 mm., and following about 0.015 sec. after the beginning of wave "a" of the venous pulse and from 0.034 to 0.046 sec. after the top of P-wave of the electrocardiogram. The average figures for the auricular sound were very similar on the different areas.

The first heart sound had a rather complicated appearance, but upon careful analysis and correlation with the mechanical events a systematization was reached. Its picture was essentially the same over all the investigated areas. Very constantly it was possible to individualize four groups of vibrations forming the first sound: the first of all, formed by only one thick vibration and following immediately after the auricular vibration when these were present, began about 0.008 sec. before the top of R, lasted about 0.035 sec., and showed a vibration frequency of 28, 36, and 24 per second over the apex, mesocardiac area, or base, respectively. The second component, formed by one and a half vibrations, began after the top of R and ended always before the initiation of "c," lasted for about 0.036 to 0.045 sec., and showed a vibration frequency of about 39 per sec. The third component, formed by two vibrations, started always rather sharply, keeping very uniform relations with the beginning of the "c" wave of the venous pulse, thus allowing the assumption that they both are due to the same cause (i.e., the opening of the sigmoid valves and initiation of the ejection phase); it lasts for about 0.050 sec. and shows

a frequency of about 39 per sec. The fourth component is formed by an average of two vibrations not so distinctly separated from those of the preceding group; it starts a short interval before the top of the "c" wave, lasts for about 0.060 sec., and shows a frequency of about 32 vibrations per second. The second and third components have the largest amplitude.

All this apparent complexity may be easily and satisfactorily explained if we assume that in the origin of the first sound two acoustic processes take place in each ventricle: one caused by the sudden contraction of the ventricles and the other by the opening of the sigmoid valves. The term "isometric component" is suggested for the first and the term "ejective component," for the second. Each one of them is modified by accessory influences. The isometric component is affected in its earliest portion by tardy auricular vibrations and by the ventricular impact on the chest wall. The ejective component is modified in its latter portion due to the acceleration of blood during the maximal ejection phase. The combined influence of all this accounts for the registration of the four components just analyzed. If sometimes the first sound shows a more homogeneous character, this is due to a more immediate sequence between two or more groups.

The second sound, although very much simpler than the first one, showed also a rather complex picture. This, however, we have been unable to analyze because, in the absence of the central arterial pulse record, we had not enough reference points to correlate the groups of vibration with the mechanical events. It lasted for 0.094, 0.116, and 0.120 sec. over the apex, mesocardiac area, and base, respectively, and showed a frequency of 36 vibrations per second all over the precordial region. It was recorded with maximal amplitude on the mesocardiac area and preceded by a uniform interval of 0.110 sec. the top of the "v" wave on all the explored areas.

The so-called third heart sound was recorded in 65 per cent of the cases, the average figures for its properties being very uniform over all the explored areas. They were for the apex, mesocardiac area, and base, respectively: duration, 0.117, 0.136 and 0.092 sec.; number of vibrations, 3, 7, 4.7, and 3; frequency, 32, 34, and 33 vibrations per second. It was always recorded after the top of "v" wave and a short interval before the end of its descending limb, consequently, during the final moments of the rapid ventricular inflow.

AUTHOR.

de Chatel, A., and Hussey, R.: Experimental Investigation of the Electrocardiogram With Esophageal Leads. Ztschr. f. klin. Med. **131:** 450, 1937.

P-waves in dogs with the chest opened are bigger in esophageal leads than in any other indirect lead. Partial block between the auricles produced by tying the interauricular band alters the form of the P-wave in esophageal leads and leads from the left chest wall much more than those from the right chest wall. The authors believe that esophageal leads are valuable clinically in diagnosing rhythm and conduction disturbances of the auricles.

KATZ.

Holzmann, M.: Clinical Observations With Electrocardiographic Chest Leads. Arch. f. Kreislaufforsch. **1:** 2, 1937.

This extensive article covering 170 pages contains a detailed description of the experiences of the author with chest leads. He is cognizant of the American literature and confirms in general the views now held in this country. He concludes that there is a definite place for this lead in that it gives evidence of abnormalities when none are present in the limb leads and adds further information when the limb leads are abnormal.

KATZ.

Körner, F.: The Cross Striations of Heart Muscle. Arch. f. Kreislaufforsch. 1: 358, 1937.

This is a review of the meaning of the intimate anatomy of the heart muscle. This includes observations of the author which show that histologically the cross striations depict the state of contraction or relaxation of the muscle fiber. In a single field the cross striations are clearly narrower and stain red in acid alizarin blue-Mallory, whereas in the relaxed fibers they are thicker and more differentiated and stain blue in the dye. Closer examination shows that this cross striation is in reality portions of fibrils which run the length of the fibers and from one segment to the next.

KATZ.

Master, Arthur M., Jaffe, Harry L., and Dack, Simon: The Heart in Acute Nephritis. Arch. Int. Med. 60: 1016, 1937.

Acute glomerulonephritis is sometimes associated with clinical symptoms of failure of the left ventricle, such as dyspnea, cyanosis, and pulmonary edema. These appear early in the course of the disease; in fact, they may be the presenting symptoms and may occur before there is evidence of renal injury. The involvement of the heart is the result of vascular and not renal damage.

The diffuse vascular change also produces hypertension, which is almost always present in the first week or two of the disease.

Changes occur in the electrocardiogram which indicate myocardial damage, that is, definite abnormalities of the T-wave in Leads I, II, and IV, absence of the initial positive deflection in the precordial lead, and prolongation of the auriculoventricular conduction time.

Acute glomerulonephritis is a systemic vascular disease in which the heart may be seriously damaged.

AUTHOR.

Berliner, Kurt, and Master, Arthur M.: Mitral Stenosis: A Correlation of Electrocardiographic and Pathologic Observations. Arch. Int. Med. 61: 39, 1938.

Reports of 113 fatal cases of rheumatic disease of the mitral valve with autopsy records were collected, and the electrocardiograms were analyzed. Associated lesions of other valves were found to be the most important single factor affecting the electrocardiograms.

Notching of the P-wave was found to be the principal electrocardiographic sign of mitral stenosis. Marked increase in height and width of the P-wave, however, was always associated with hypertrophy of both auricles and was therefore found to be common only in cases of mitral stenosis associated with disease of the tricuspid valve, and in these cases the notching was generally more marked.

Right ventricular preponderance was noted in less than half the cases of uncomplicated disease of the mitral valve and therefore cannot be regarded as a characteristic sign of mitral stenosis. Right ventricular preponderance, however, was generally found in the "button-hole" type of mitral stenosis, but still more frequently right ventricular preponderance was due to an associated lesion of the tricuspid valve.

Left ventricular preponderance was never found in any case of mitral stenosis unless disease of the aortic valve also was present. When mitral stenosis was associated with aortic insufficiency, electrocardiographic signs of ventricular preponderance depended solely on the extent of the leak in the aortic valve; in all cases of marked aortic insufficiency with high pulse pressure left ventricular preponderance was present, whether the associated mitral stenosis was slight or marked.

The voltage of the chief ventricular deflection (QRS) in cases of mitral stenosis was never above normal unless aortic insufficiency coexisted. In cases of mitral stenosis with atrophy of the left ventricle, the voltage of QRS was normal.

The electrocardiograms of persons with pure mitral insufficiency without stenosis were distinguished from those of persons with mitral stenosis by a normal or nearly normal P-wave. Auricular fibrillation or auricular flutter never occurred in pure mitral insufficiency, and ventricular preponderance was never to the right.

Complete change from right ventricular preponderance to left ventricular preponderance and vice versa occurred only in cases of mitral stenosis associated with lesions of both the tricuspid and the aortic valve.

A correlation of the post-mortem observations and the electrocardiograms revealed that the electrocardiographic signs of ventricular preponderance, when present, indicated the anatomic relationship of the ventricles correctly in 89 per cent of the cases.

AUTHOR.

Bredt, H.: Can Congenital Heart Disease Cause Morphological Changes in the Pulmonary Circuit? Klin. Wchnschr. **15**: 1358, 1936.

Congenital heart disease by leading to hypertension in the pulmonary circuit can cause a secondary pulmonary sclerosis which does not involve the smallest branches.

KATZ.

Kreuzfuchs, S.: The Course of the Aorta and Its Measurements in Childhood. Fortschr. a. d. Geb. d. Röntgenstrahlen. **54**: 396, 1936.

The aortic arch in childhood differs from that of the adult. In adults the arch has a horizontal angulated course with a pretrachial-frontal and a paratracheal-sagittal division. This angular deviation is due to the action of the trachea. When the trachea is deviated to the right, the aorta loses its angle and assumes a diagonal course and is arched as usually depicted anatomically. In childhood the trachea is to the right, and only during puberty does it slowly deviate toward the mid-line. In childhood, therefore, the aorta is arched and diagonal in position.

The adult aortic type gives a characteristic x-ray double limb shadow with intensification of the aortic knob. The infantile aortic type gives a single limb shadow, eliptical in shape. In childhood the aorta must be measured in Fechter's position.

In this way the author found values of the aorta as follows:

10 mm. at 5 years
12 mm. at 10 years
14 mm. at 12 years
16 mm. at 14 years
20 mm. at 20 years

Occasionally the infantile aorta persists into adult life.

KATZ.

Ash, Rachel: Influence of Tonsillectomy on Rheumatic Infection. Am. J. Dis. Child. **55**: 63, 1938.

An analysis is made of the effect of tonsillectomy on the course of rheumatic infection in 522 children treated at the Children's Hospital between 1922 and 1936.

Tonsillectomy did not prevent recurrences of rheumatic manifestations.

Neither the presence or the absence of tonsils at the time of the initial infection nor the removal of tonsils subsequent to the onset had any demonstrable influence on the incidence of cardiac involvement or the death rate, due consideration being paid to the associated variable statistical factors.

A high incidence of rheumatic exacerbations followed immediately on tonsillectomy performed early in the course of the disease.

Tonsillectomy for the rheumatic child seems to be indicated only when there is definite evidence of disease in the tonsils and not as a routine procedure. The operation should be performed only during an inactive phase of the infection. Associated with the absence of other symptoms, the combination of a normal temperature curve and a normal sedimentation rate of the erythrocytes may be taken as indications of an inactive phase.

AUTHOR.

Cochems, K. D., and Kemp, J. E.: Occupation and Syphilitic Aortitis. Am. J. Syph. Gonor. & Ven. Dis. **21**: 408, 1937.

Seven hundred forty-nine male individuals, predominantly white, in all stages of syphilitic infection, and about whom all the necessary data were available, were analyzed according to the physical strain consequent to their occupation and the incidence of syphilitic aortitis. This analysis showed:

The incidence of syphilitic aortitis was greater among individuals of intermediate and heavy occupational pursuits (14.4 per cent) than it was among individuals following sedentary occupations (8.7 per cent).

The incidence of uncomplicated syphilitic aortitis was greater among individuals following the lighter occupations (5 per cent) than it was among those whose work was more strenuous (3.7 per cent).

Aneurysm occurred four times as frequently among individuals following occupations demanding a certain amount of physical exertion (6 per cent) than among those whose occupations were sedentary (1.4 per cent).

The incidence of aortic insufficiency was higher among individuals doing heavy labor (2.1 per cent) than among those whose occupations were light (0.7 per cent).

The incidence of aneurysm and aortic insufficiency occurring together was not influenced by the physical stress consequent to occupation.

MONTGOMERY.

Ernstene, A. Carlton: The Cardiovascular Complications of Hyperthyroidism. Am. J. M. Sc. **195**: 248, 1938.

A study has been made of the cardiovascular complications in 1,000 consecutive cases of hyperthyroidism.

Clinical or electrocardiographic evidence of organic heart disease was present in 173 patients; 32 others had enlargement of the heart by roentgenologic examination but no other signs of organic heart disease.

Auricular fibrillation occurred in 207 patients. In 96 of these, the arrhythmia was present before operation either in its continuous form or in paroxysms of variable duration, while in 111 it developed for the first time as a postoperative complication.

The factors which influenced the incidence of auricular fibrillation most importantly were the presence of organic heart disease, the age of the patient, the duration of the hyperthyroidism, and the type of goiter. The degree of elevation of the basal metabolic rate had no effect.

Normal sinus rhythm was reestablished spontaneously in one-third of the patients with the continuous form of auricular fibrillation. Quinidine sulphate was administered to one-half of the remaining patients in this group, and in 60 per cent of these reversion to sinus rhythm occurred.

Congestive heart failure was present in 44 patients. The two most important factors responsible for this complication were organic heart disease and uncontrolled auricular fibrillation.

The postoperative mortality was considerably greater in patients who had auricular fibrillation either before or after operation than it was in individuals with normal cardiac rhythm.

AUTHOR.

Veal, J. Ross: The Management of Arteriosclerotic Disease of the Lower Extremities. South. M. J. 31: 54, 1938.

The outstanding considerations of such management are:

1. The disease progresses by definite stages.
2. Early recognition and prompt treatment will relieve many patients and restore them to a useful and at least a partially active life.
3. Gangrene can frequently be prevented by cooperation between physician and patient and the consistent use of simple prophylactic measures.
4. When once gangrene has developed, and its development is inevitable in some cases, reduction of the surgical mortality can be achieved in three ways: (a) By careful preoperative preparation, based on the conception of arteriosclerotic gangrene as a systemic condition first and a local condition second. (b) By all the precautions usually taken to avoid trauma, shock, and hemorrhage at operation plus, in our experience, the use of the circular amputation in practically all cases. (c) By proper postoperative therapy.

MONTGOMERY.

Goldblatt, Harry: Studies on Experimental Hypertension. V. The Pathogenesis of Experimental Hypertension Due to Renal Ischemia. Ann. Int. Med. 11: 69, 1937.

Persistent hypertension has been produced in dogs and monkeys by constricting the main renal artery with a special silver clamp. In some of the dogs the hypertension has persisted for more than five years. The type of hypertension produced depends upon the degree of constriction of the renal arteries. Constriction of splenic and femoral vessels and of the superior mesenteric artery has produced no effect on the blood pressure. A number of experiments have been performed in an attempt to explain the mechanism of the production of the hypertension. In six dogs, one or both clamps were released or removed some time after hypertension had developed, with prompt restoration of the blood pressure to the original levels. In three dogs removal of the ischemic kidney also was followed by prompt return of the blood pressure to the original levels. Bilateral nephrectomy did not produce hypertension. Bilateral adrenalectomy with or without supportive or substitution therapy prevented the development of the hypertension or interfered with the maintenance of the hypertension which followed the renal ischemia. The results of these and other experiments indicate that this type of experimental hypertension is due mainly to a humoral mechanism and that the adrenal cortical hormone plays an important part in the production or maintenance of the hypertension.

HINES.

Pilcher, Robin: Pulmonary Embolism. A Statistical Study of Its Incidence in Twelve London Hospitals in the Decade 1925-34. Brit. J. Surg. 25: 42, 1937.

There were 731 cases of fatal pulmonary embolism proved by post-mortem examination. This is 0.105 per cent of all surgical deaths; 0.064 per cent of all medical deaths. The true incidence of pulmonary embolism must, of course, be higher than this. In 473 of the 731 cases a search was made for the site of primary thrombosis, and in 362 evidence in peripheral vessels was found.

MONTGOMERY.

Medical Progress. Clin. J. 66: 345, 1937.

Embolism After the Injection of Varicose Veins.

Westerborn in Sweden analyzed the cases of some 20,000 patients who were given injections for varicose veins. There were 11 deaths from pulmonary embolism. In 6 other cases signs of severe embolism were followed by recovery. More radical

surgical procedures on varicose veins have a much higher mortality. Westerborn found that 6,994 operations on varicose veins were followed by 18 deaths from embolism. In America McPheeters found that 6,671 operations on varicose veins were followed by 36 deaths from embolism—a mortality from embolism of 0.54 per cent.

MONTGOMERY.

Homans, John: Venous Thrombosis in the Lower Limbs: Its Relation to Pulmonary Embolism. Am. J. Surg. **38**: 316, 1937.

The influences, anatomic and related to life in bed, consequent upon many diseases, injuries, and operations, which lead to thrombosis in the lower half of the body, are fairly well recognized today. Many of them are unavoidable, but most of them can be relieved of some of their bad effects by prophylactic treatment.

Thrombosis once established should no longer be treated by the familiar ice bag and immobilization but by real elevation followed early by gradually increasing exercise. Embolism is more likely to be prevented by forestalling the formation of the dangerous propagating thrombus than by apprehensive immobilization. A further study of deep peripheral thrombophlebitis should be made by both the physicians and pathologists. By such means the apparent tendency today to increase in the incidence of thrombophlebitis may be reversed.

AUTHOR.

Barnes, A. R.: Pulmonary Embolism. J. A. M. A. **109**: 1347, 1937.

Death from pulmonary embolism is a much greater menace in both medical and surgical cases than is generally realized. Although its cause is not known, some of the factors that predispose to its occurrence are known. Mild premonitory attacks frequently precede the fatal seizure, and it is important that they be recognized. The picture of shock, noted as much as or more commonly than marked dyspnea and cyanosis, may constitute the clinical symptoms of pulmonary embolism. The electrocardiogram may furnish invaluable aid in the diagnosis of this condition and especially in its differential diagnosis from acute coronary thrombosis. Whatever the cause of pulmonary embolism, the most promising avenue of attack is the attempt to improve the rate of circulation and particularly the velocity of venous return from the lower extremities. A comprehensive program looking to that end should be applied, if not to all patients, then to those patients whose condition presents circumstances which are known to predispose to the occurrence of pulmonary embolism. Certain results to date encourage one to believe that if such a program were carried out with uncompromising zeal a very high percentage of deaths from pulmonary embolism could be eliminated, at least following surgical procedures, during the puerperium, and following sprains and fractures. In no aspect of surgery is there such a promising field for lowering surgical mortality. The medical profession is challenged to use at least such measures as are available in the effort to combat the tragic situation presented by pulmonary embolism.

AUTHOR.

Johnston, C. H.: Combined Ligation and Injection Treatment of the Varicosed Great Saphenous Vein. J. A. M. A. **109**: 1359, 1937.

Recurrence in cases presenting incompetent saphenofemoral valves when treated by injection alone or by ligation alone is far too common. Ligation at the saphenofemoral junction, dissecting out, and section of all five branches at that level, and injection of the distal end of the saphenous vein, is the treatment of choice. All

cases in which the saphenofemoral valves are incompetent are indicated for the ligation-injection treatment. Until some newer idea or operations supplants the ligation-injection form of treatment, it must be admitted that it gives the greatest promise of permanent success with the least amount of danger, pain, or mutilation in the more extensive varicose veins.

AUTHOR.

Bird, Clarence E.: The Use of Arteriography of Substitutes for Colloidal Thorium Dioxide. J. A. M. A. **109**: 1626, 1937.

Because of the possibility that the injection of radioactive thorium dioxide solution may cause late toxic symptoms, other materials for arteriography are preferable. Diodrast, neoskiodan, uroselectan and similar preparations made for excretory urography are nontoxic in the doses used and are quickly excreted. They do not damage the intima of the vessels and do not cause pain on intravascular injection (as does sodium iodide). The density of the shadow cast by diodrast and similar iodine-containing solutions when used for arteriography is not quite so striking as that by thorium dioxide, but is entirely satisfactory.

Accompanying photographs of x-rays of arteriovenous aneurysms show the aneurysms clearly and outline many small arteries.

MONTGOMERY.

Koller, S.: The Mortality of Circulatory and Respiratory Diseases. Arch. f. Kreislaufforsch. **1**: 225, 1937.

The author presents a statistical assay of 1,600,000 deaths of circulatory origin occurring in England and Wales from 1921 to 1933. He is able to show a seasonal variation in death rate with the peak in winter and the low point in summer.

KATZ.

Holzlöhner, E.: The Respiratory Pulse in Man and the Blood Flow in the Veins Near the Heart. Arch. f. Kreislaufforsch. **1**: 305, 1937.

This is a comprehensive review of the subject with a detailed presentation of data to demonstrate that the heart itself causes a systolic acceleration of blood flow in the veins near the heart. The result is that there is an early filling of the auricle and the ventricle independent of diastolic duration. Changes in systolic power of the heart by altering systolic acceleration of blood flow will in this way alter the filling of the heart. An automatic regulation of filling dependent on the power of ventricular systole is thus provided. This systolic venous acceleration is in reality a form of useful work of the heart. Pneumothorax abolishes this systolic venous acceleration. It is probably modified also in incompetence of the A-V valves. These considerations suggest a clinical utility in recording the respiratory pulse. The author describes a string anemometer which he has used for this purpose. This consists of a small wire suspension in an air passage placed in the optical axis of an illuminated microscope system much as is used in the string galvanometer. The air passage is connected to the nasal passage by tubing as in the hot wire arrangement. This meter can be calibrated.

KATZ.

Ludwig, H.: The Heart Kymogram. Fortschr. f. Röntgenstrahlen. **54**: 469, 1936.

The author points out that in the roentgenokymogram not only local pulsations were obtained but also rotations and displacements of the whole heart. For this reason care must be taken not to read too much into the curves obtained.

KATZ.

Ludwig, H.: Auricular Flutter in the Kymogram. Röntgenpraxis 8: 731, 1936.

In this case it was found that flutter waves of the right auricle and left auricle could be distinguished in the roentgenokymogram. These corresponded in frequency with those seen in the electrocardiogram.

KATZ.

Trumble, Hugh G.: Strategic Points in the Lumbar and Sacral Outflows of the Autonomic System: Sympathetic Denervation of the Lower Limbs. M. J. Australia 2: 958, 1937.

The technique is a muscle-splitting abdominal operation without opening of the peritoneum.

The whole operation is performed in twenty minutes or less. The patient may be allowed to sit up out of bed on the fourth or fifth day postoperatively. The author has always observed a persistent increase of the temperature of the skin of the leg.

MONTGOMERY.

Barker, M. H.: The Use of Cyanates in the Treatment of Hypertension. Wisconsin M. J. 36: 28, 1937.

The cyanates have been administered over a period of seven years to patients who have been under controlled observation for several weeks or months. Symptomatic variations, blood pressure effects and certain blood chemical observations have been correlated with the concentration of the cyanates in the blood. Taking the group as a whole, slightly more than 50 per cent have shown significant decreases in both systolic and diastolic blood pressure, and these drops of pressure were associated with the elevation of the cyanates in the blood to 6-12 milligrams per 100 c.c. Doses of 0.3 gram of potassium sulfocyanate two to fourteen times per week indicate the individual requirement for constant therapeutic blood concentration.

The best symptomatic and pressure-lowering results were obtained in the menopausal and essential types. The malignant forms were rarely benefited. Occasional hypertensive patients with arteriosclerosis showed good improvement, but many suffered only toxic manifestations. A few cases of chronic nephritis showed satisfactory drops in blood pressure, and an occasional diuresis was noted. Toxic manifestations of fatigue, weakness, mental confusion, disorientation, and nausea were encountered. As a rule, only fatigue was noted and chiefly in arteriosclerotic individuals. No dangerous symptoms or findings were met unless the blood cyanates rose over 20 milligrams.

AUTHOR.

Book Reviews

ORTHODIASCOPY—AN ANALYSIS OF OVER SEVENTEEN HUNDRED ORTHODIASCOPIC EXAMINATIONS. By Chester M. Kurtz, M.D., F.A.C.P., Assistant Professor of Medicine, University of Wisconsin; Cardiologist to the State of Wisconsin General Hospital. New York, 1937, The Macmillan Company. Price $3.50.

This monograph emphasizes the value of a quantitative determination of the size of the abnormal heart in diastole by means of the orthodiagram. The author presents a detailed and analytical consideration of 1,723 cases on whom mensuration of the frontal plane of the heart by this method is compared with the formula for predicting the normal frontal area (Method of Hodges and Eyster) on the basis of age, height, and weight of the individual concerned. To this there is added the greatest transverse diameter of the cardiac silhouette which is compared with normal figures based on similar prediction tables. From these observations the author has concluded that in patients with evidence of cardiac enlargement to physical examination the deviation of the frontal area is almost invariably above the level of 10 per cent, a figure which, expressed in percentage deviation, is abnormal. The cardiothoracic ratio, used so extensively at present as a sign of enlargement of the heart, was found "worthless."

As the author well states, this method is only an additional aid in indicating the probability of the diagnosis in question being correct or incorrect. It is a pleasure to see him revert back to clinical impressions whenever he is in doubt, despite the detailed measurements obtained, especially so in patients with so-called "uncomplicated" mitral stenosis and in mitral insufficiency.

The book is well written and printed in large type. It is a worthy addition to our literature on diseases of the heart and merits the attention of all those who are not as yet acquainted with these methods of mensuration.

A MONOGRAPH ON VEINS. By Kenneth J. Franklin, D.M., M.R.C.P. Tutor and Lecturer in Physiology, Oriel College. University Demonstrator of Pharmacology. Assistant Director of the Nuffield Institute for Medical Research, Oxford. Springfield, Ill., Baltimore, Md., 1937, price $6.00, Charles C. Thomas.

The timeliness of this volume of some 400 pages will not be questioned by anyone at all familiar with the recent astonishing growth of interest in the study of the peripheral circulation.

Although comparatively little space is given to a consideration of the clinical aspects of disorders of the veins the book is a mine of information concerning the embryological, anatomical, physiological, and pharmacological facts and theories upon which the clinical study of the subject should be based.

The mere listing of the headings of the more important chapters will perhaps serve to give an idea of the character and scope of the monograph—"The Embryology of Veins"; "The Anatomy of the Venous System, Functionally Considered"; "The Valves in Veins"; "The Amount of Circulating Blood: Blood Depôts"; "Comparative Anatomy"; "The Venules"; "Absorption by Veins and Diffusion From Veins"; "Veins and the Nervous System" (three chapters); "The Heart and the Venous Return"; "The Effects of Hydrostatic Pressure"; "The Effects of the Contraction of Voluntary Muscle Upon Venous Return"; "Respiration and the Venous Return in Mammals; the 'Vis a Fronte'"; "Venous Pressure";

"The Movements of the Blood in Veins"; "Clinical." One of the last chapters is devoted to a discussion of the various photographic techniques applicable to research on the venous system.

The book is freely illustrated and has a very extensive bibliography. In the opinion of the reviewer it constitutes a major contribution to the study of the circulation, both central and peripheral.

PATHOLOGIE DES KREISLAUFS. Ein Lehrbuch der Herz und Gefässkrankheiten von Prof. Dr. Theodor Brugsch, Berlin. Professor der Medizin und Direktor a. D. der Med. Universitätsklinik Halle-Wittenberg. 92 illustrations, 2nd edition, Leipzig, 1937, price, RM 30, S. Hirzel.

The scope of this book of 747 pages is wider than the general reader is likely to associate with its title, the "Pathology of the Circulation." It deals with the disorders of the heart, the peripheral blood vessels, and the general circulation, and few items concerning these have escaped at least a brief consideration, ranging from rare congenital anomalies of the heart to varicose veins of the extremities. More than five-sixths of the text is devoted to matters pertaining to the heart directly, the remainder to the peripheral vessels. The first, or the analytical, portion of the book contains sections on the normal anatomy and physiology of the various components of the cardiovascular system, on cardiovascular symptoms and signs, on the mechanism of the heart sounds, murmurs, pulse, arterial and venous pressures, and on special techniques employed in diagnosis. The space allotted to arterial and venous sphygmograms which are now rarely used in clinical diagnosis might have been more profitably used to amplify the sections on fluoroscopy and electrocardiography, especially the latter, the treatment of which does not seem adequate (9 pages with very few illustrative records). The first 160 pages of material are presented as the groundwork for the comprehension of the numerous clinical varieties of cardiovascular disorders to which the remainder of the book is devoted, namely, various forms of vascular collapse, cardiac insufficiency, the arrhythmias, valvular disease, coronary disease, and such other sections as are found in standard textbooks on heart and vascular diseases. The section on "Constitution and Circulation" is well worth reading.

The manner of treatment of some of the material reveals conflicting influences. On the one hand, the author stresses the need of evaluating the status of a cardiac patient, not only by a structural, a physiological, or a functional derangement, but by all of them considered together in the light of the constitutional type to which the individual belongs. Some of the sections, however, disclose a strong influence by an opposite point of view when extensive discussion is given not only of the diagnosis but of the prognosis and treatment of valvular lesions. Consideration of cardiac diseases in this fashion serves to obscure the fact that the valve damage is only an element in a disease and not the disease itself. The study of the natural history of heart diseases in recent years, especially that of rheumatic fever, in which the object of attention is not so much the evolution of an endocardial or myocardial injury, but the life and activity of the subject, has thrown considerable light on prognosis and treatment. One misses such discussions of natural history in this book. Except for an occasional reference, there is no bibliography.

The quality of the material is uneven. The sections on treatment are perhaps, on the whole, the weakest. The 38 pages devoted to digitalis presents an unusual number of generally abandoned theories, statements without adequate support, and specific therapeutic recommendations without sound basis in theory or practice. A few of these may be mentioned by way of illustration. Chronic cardiac insufficiency, the author maintains, is abolished by 1½ gr. of digitalis daily for ten days. In his method of using large doses for the treatment of severe heart failure he recommends 6 gr. daily for two days, 4½ gr. for two days, and 3 gr. for three days, and

warns against such large (?) doses if the patient has received digitalis during the previous week. In the use of this method, he maintains, the infusion of digitalis is the most suitable preparation in the first two to three days. He states that if, during the course of left heart failure, the right heart also fails, causing general venous stasis, intravenous strophanthin is preferable to digitalis. He states that digitalis dilates the renal vessels and perhaps also stimulates the renal parenchyma, and that the proprietary preparation, digipurat, has a greater action on the kidney. He strongly recommends intravenous strophanthin therapy and states that in decompensation due to valvular diseases the dose depends upon the valve involved: 0.5 mg. strophanthin in mitral insufficiency, 0.4 mg. in aortic insufficiency, 0.2 to 0.3 mg. in mitral stenosis. He states that a brief course of digitalis will restore compensation which has failed as the result of valvular disease, whereas in "pure" cardiac insufficiency the continued use of digitalis is necessary. To counteract the excessive slowing by digitalis he recommends atropine and thyroid, and to counteract coronary constriction he recommends a mixture of caffeine, theobromine, and papaverine. Such thoughts, presented, as they are, without evidence, will for the most part seem strange and unattractive to the critical student of cardiovascular disease.

The style of the book is fairly direct and usually interesting. Many of the discussions are thought-provoking. The author's inclination to rather liberal speculation, however, makes this book safer in the hands of the critical and experienced cardiologist than in those of the average student or practitioner for whom the author states the book is intended.

MACLEOD'S PHYSIOLOGY IN MODERN MEDICINE, ed. 8, edited by Philip Bard. St. Louis, 1938, The C. V. Mosby Company.

The section on the circulation, covering 226 pages, was written by H. C. Bazett. The amount of material presented is extremely large, and the descriptions are very detailed. This textbook of physiology contains information concerning the circulation which is larger in amount, more detailed, and more up-to-date than that which is to be found in any other book with which this reviewer is acquainted. A large bibliography is provided. The sections on cardiac output, on blood velocity, on pulse wave velocity, and on the reactions of cutaneous vessels are very well done, and the value of the newer data is assessed from a point of view possible only to an active investigator. A section on hemorrhage, oligemia, plethora, shock, and cardiac abnormalities is written from a viewpoint which shows that the author has not lost his interest in clinical medicine.

The presentation differs from that in most textbooks in that it places more emphasis on the physical point of view. This will make certain sections hard reading for those who are unfamiliar with physical and mathematical conceptions, as most practicing physicians are. The diction might be clearer. Its great value will be as a book of reference for clinicians who have no time to read the ever increasing bulk of original physiologic literature.

ISAAC STARR.

LEHRBUCH DER ELEKTROKARDIOGRAPHIE. von Dr. D. Scherf. Privatdozent für Innere Medizin an der Universität Wien. 169 text illustrations, Vienna, 1937, price RM 16.50, Julius Springer.

Dr. Scherf has written a very serviceable book in "Lehrbuch Der Elektrokardiographie." The plan of the book follows the conventional method. It begins with the general principles of electrocardiography, the apparatus and description of the normal electrocardiogram and its variations. Then follows a section describing abnormalities of the form of the electrocardiogram, correlating diseases with the electrocardiographic changes in a very satisfactory fashion. He follows the middle

ground of presenting the conventional or "old terminology" of bundle-branch block, but also gives the "new terminology." In the section having to do with coronary occlusion and coronary artery disease, precordial chest leads are discussed. The section which is given over to the irregularities is very well done, except for those parts having to do with treatment. These appear to me to be inadequate and not especially judicious, probably because the plans of therapy do not coincide with my own notions of the use of the measures and drugs which are available. The use of illustrations has been very generous. The bibliography has not been selected with as much care as the rest of the book deserves.

The volume is a very welcome addition to the library of those especially interested in electrocardiography, but it will find little place outside this group in this country since our own literature abounds in excellent texts for students and practitioners.

The appearance of a second, revised edition of the book within a few months of the printing of the first edition testifies to the popularity that it has won.

A Dissertation on Acute Pericarditis. By Oliver Wendell Holmes, M.D. January 12, 1836, 39 pp., 12 mo., Boston, The Welch Bibliophilic Society, 8 Fenway, price $7.50. Introduction by James F. Ballard, Director, Boston Medical Library.

Oliver Wendell Holmes, after having graduated from Harvard College in 1829 and having read law for a year, decided to take up the profession of medicine. After taking two courses in a private medical school in Boston he went to Paris where for two years he walked the wards of La Pitié and the Hôtel Dieu in the footsteps of two great clinicians Louis and Andral. Returning to Boston late in 1835, he was given the degree of M.D. by Harvard in 1836 and as one of the requirements for that degree offered this dissertation on pericarditis.

By some strange circumstance the manuscript of this essay has remained all this time unknown and unpublished in the archives of the Boston Medical Library and has only now seen the light of day in this publication by the Welch Bibliophilic Society.

The writer presents an analysis of the anatomical and clinical features of eleven cases observed with Andral and Louis, as well as of a larger number of cases recorded by Bouillaud, and discusses the value of the "antiphlogistic" treatment in general use at that time.

He quotes Bouillaud as stating that "of twenty patients attacked with acute general rheumatism of the articulations, there will be at least half who will offer the symptoms of pericarditis or endo-carditis (inflam. of the internal membrane of the heart) and often of both united."

The volume is an attractive, finely printed brochure of forty pages which should be warmly welcomed by medical bibliophiles as well as by all who are interested in the history of this important disease.

Books Received

Lecciones de Cardiologia. II. By J. Montes Pareja, fascículo. Montevideo, Uruguay, 1937.

Los Ruidos Cardíacos en Condiciones Normales y Patológicas. Por Oscar Orías y Eduardo Braun-Menéndez. El Ateneo, Librería Científica y Literaria. Florida 371—Sucursal: Córdoba 2099, Buenos Aires, 1937.

Lac Arritmias en Clínica. Por el Doctor Antonio Battro. Docente Libre de Clínica Medica, Jefe de Clinica Honorario de la Cátedra del Prof. Mariano R. Castex. El Ateneo, Librería Científica y Literaria. Florida 371—Sucursal: Córdoba 2099, Buenos Aires, 1937.